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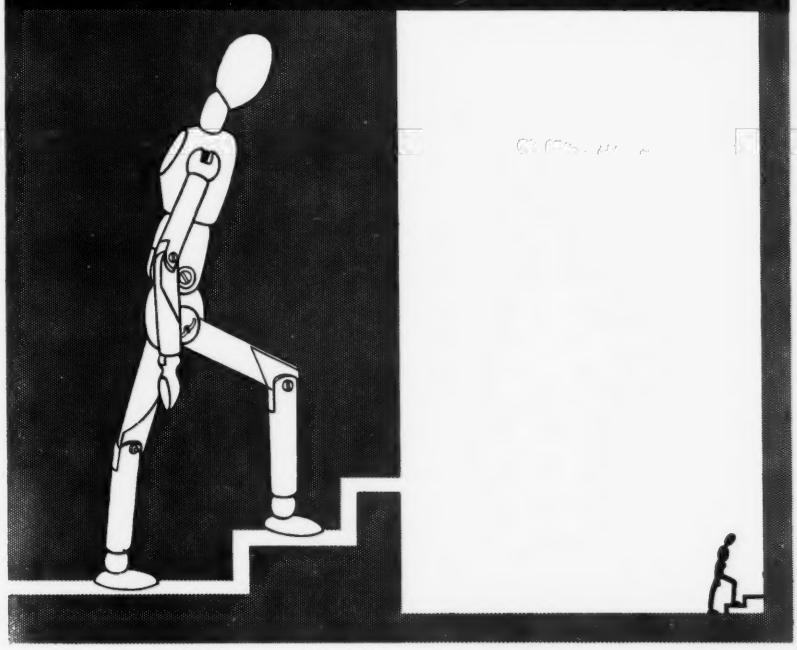

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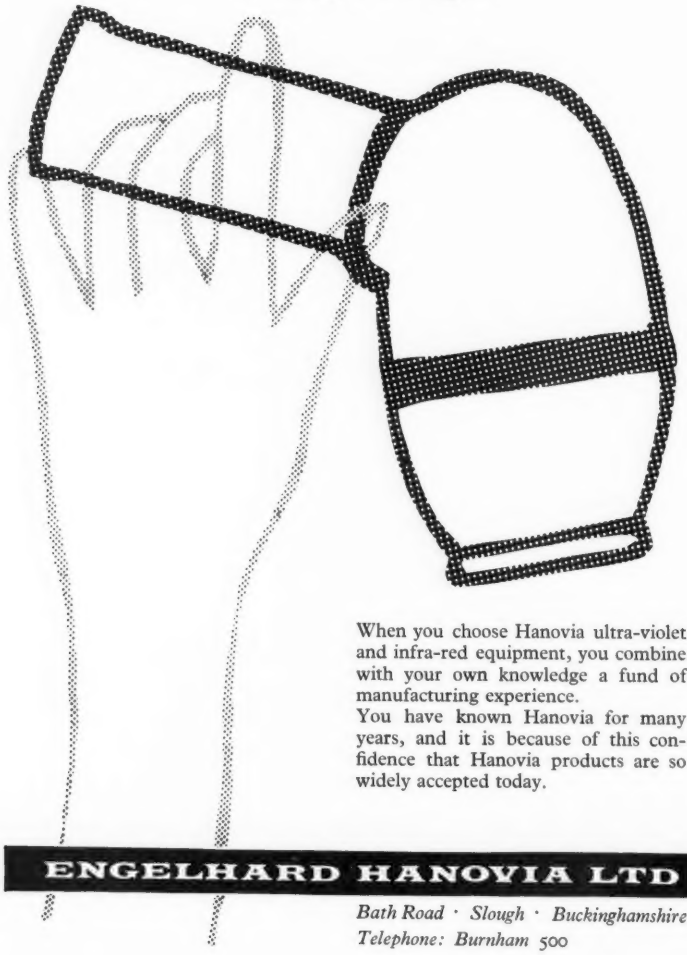

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ORIGINAL PAPER

METABOLIC DISEASES AFFECTING THE LOCOMOTOR SYSTEM

II. OSTEOPOROSIS AND OSTEOMALACIA*

BY STEPHEN MATTINGLY

From the Middlesex Hospital, London

METABOLIC bone disease may be due to inherited metabolic defects, dietary deficiencies, and gastro-intestinal, hepatic, renal, and endocrine disease. The subject is complex and controversial, and in this paper I can only discuss briefly two conditions, osteoporosis and osteomalacia, due to defective bone formation in adults.

We tend to think of bones as dried anatomical specimens, with a characteristic shape and structure determined by such stresses as weight-bearing and locomotion, and marked in a curiously uniform way by the attachments of muscles and ligaments. After death they appear almost indestructible, but during life bone is constantly being destroyed and replaced. In the young, bone formation exceeds destruction, but, in the elderly, bone is removed faster than it is manufactured.

Bone formation in adults may, for convenience, be divided into two stages: (1) osteoblasts form an organic matrix, rich in protein, called osteoid, and (2) this bone matrix is calcified.

OSTEOPOROSIS

Osteoporosis is due to defective formation of bone matrix. As a result, bone destruction exceeds bone formation and there is a slow but constant loss of

* The second paper of a symposium presented at the Annual Meeting of the British Association of Physical Medicine, April 29, 1960. Paper III will appear in our next issue.

calcium from the skeleton. After several years in negative calcium balance, X rays reveal rarefaction of bone.

Osteoporosis is the commonest metabolic disease affecting bone, but, although it may be observed in old age and can follow disuse, malnutrition, and endocrine disorders, its cause is not always evident, especially when younger patients are affected.

It seems that osteoblasts are normally stimulated by muscular activity, and immobilization of the skeleton in bed or in a plaster cast, especially when muscles are paralysed, often results in osteoporosis (Howard *et al.*, 1945; Dietrich *et al.*, 1948; Stevenson, 1952; Sissons, 1952). Since most of the body calcium is stored in the bones, excess calcium from porotic bones is excreted in the urine. If patients are suddenly paralysed by poliomyelitis or spinal injury, hypercalciuria may result in the formation of renal stones, with subsequent urinary infection. This is one good reason for encouraging exercises and frequent changes of posture when patients are confined to bed. Although the serum calcium concentration is usually normal in osteoporosis, hypercalcaemia may be an unexpected complication of immobilization and result in a mistaken diagnosis of hyperparathyroidism (Albright and Reifenstein, 1948; Mason, 1957).

Disuse may be partly responsible for the osteoporosis of rheumatoid disease or that encountered in elderly patients. Radiographs of patients immobilized by chronic arthritis sometimes reveal an unexpected and severe degree of bone atrophy (Plate XXI, Fig. 1).

Malnutrition is likely to cause osteomalacia rather than osteoporosis, but protein starvation and lack of vitamin C can interfere with the formation of bone matrix in the experimental animal (Bourne, 1956). Some workers believe that osteoporosis is commonly due to inadequate intake of minerals and to a persistent negative calcium balance (Nordin, 1959).

Lack of oestrogens appears to be an important cause of osteoporosis in women after the menopause (Albright *et al.*, 1941), and the osteoporosis which develops in old age and sometimes occurs in acromegaly, in eunuchs, and in gonadal dysgenesis is also attributed to lack of sex hormones. While it is clear, however, from numerous experiments on animals and birds that deficiencies of oestrogens and androgens can delay the ossification of growing bones, the effect varies from one species to another, and adult animals are not noticeably affected (Silberberg and Silberberg, 1956). It has certainly been shown that large doses of these hormones can improve the calcium balance of patients with osteoporosis (Albright and Reifenstein, 1948), and it is claimed that long-term treatment with much smaller doses is effective (Cooke, 1955). However, even if stilboestrol is only given in daily doses of 0·5–1 mg. it may cause metropathia in women, and

treatment must be discontinued every few weeks to allow for withdrawal bleeding. Methyl testosterone in a daily dose of 20–30 mg. may cause acne, hirsutism, and an increase in libido in women, while both androgens and oestrogens can cause oedema in both sexes. A cheap and reliable anabolic hormone without these side-effects would be invaluable.

Patients suffering from thyrotoxicosis sometimes develop osteoporosis, and this has been attributed to lack of available protein. However, Wayne (1960) has recently suggested that it is due to a persistent negative calcium balance, and claims that the excessive excretion of calcium in the urine is abolished with antithyroid drugs. However, Follis (1953), in a post-mortem study of 20 patients with thyrotoxicosis and "osteoporosis", found the histological changes typical of hyperparathyroidism in all 20 patients, those of osteomalacia in 6, and of osteoporosis in only 4. Unfortunately no biochemical or radiological findings were available. It seems that thyrotoxic patients may develop porotic bones, but this does not necessarily indicate that they have osteoporosis.

It is well known that some patients with Cushing's syndrome develop osteoporosis, and patients treated with adrenal steroids frequently fracture vertebral bodies, and occasionally other bones such as the femur or ribs. This may be due to the direct action of adrenal steroids on bone matrix, but they may also affect bone formation indirectly. There is some evidence, for example, that steroids enhance the absorption of calcium in steatorrhoea and in hypoparathyroidism (Hodgkinson and McCracken, 1959), although, on the contrary, they are reported to have an anti-vitamin-D effect in sarcoidosis (Anderson *et al.*, 1954; McSwiney and Mills, 1956).

Finally, a severe and generalized osteoporosis sometimes develops in younger patients without any demonstrable cause and is very resistant to treatment (Jackson, 1958) (Plate XXI, Fig. 2). It may be difficult to distinguish this "idiopathic osteoporosis" from osteogenesis imperfecta, and these patients do not necessarily have the same disease.

While the symptoms and signs of osteoporosis are often confined to the spine, they may be very similar to those which I shall describe for osteomalacia. This condition must be excluded by appropriate biochemical tests, for, with the exception of hypercalciuria, the results of these tests are usually normal in osteoporosis.

OSTEOMALACIA

Osteomalacia, or adult rickets, is due to defective calcification of newly formed osteoid. Although it is unlikely that bone matrix is calcified simply by the precipitation of insoluble calcium and phosphate salts, as Albright and

Reifenstein (1948) suggest, the concentration of calcium and phosphate ions is reduced in the blood and tissue fluids of patients with osteomalacia. This may be due to: (1) a diet deficient in calcium and vitamin D; (2) defective absorption of calcium and vitamin D from the small intestine; (3) excessive excretion of calcium or phosphate in the urine; and (4) "vitamin-D resistance".

An inadequate diet is a common cause of osteomalacia in the Far East, but is rare in Britain, though food cranks and elderly people may live on diets which are deficient in minerals and vitamins.

Vitamin-D deficiency may be due to defective fat absorption in sprue, coeliac disease, idiopathic steatorrhoea, chronic pancreatic disease, Whipple's disease, chronic liver disease, and after gastrectomy (Pyrah and Smith, 1956; Mellick, 1959).

Calcium absorption is impaired by lack of vitamin D and by the formation of insoluble calcium salts in the small intestine in the presence of excess fatty acids, or of phosphates in chronic renal failure.

The majority of patients suffering from osteomalacia in Great Britain have steatorrhoea, though this may be symptomless. Some of these patients respond to treatment with small physiological doses of vitamin D (e.g., 0·25 mg. =10,000 i.u. daily), but many require much larger doses (e.g., 5–15 mg.= 200,000–600,000 i.u. daily) even when these are given parenterally. They are said to have "vitamin-D resistance".

Patients in chronic renal failure may develop renal osteodystrophy, a mixture of osteomalacia and secondary hyperparathyroidism, and another group of patients with "vitamin-D-resistant" osteomalacia have in common a disordered function of the renal tubules, usually without much glomerular damage. Most, if not all, fail to reabsorb phosphates from the tubules, and in addition may fail to reabsorb sugar, amino-acids, potassium, or water, or to acidify the urine, with resultant loss of base. Such patients, with renal tubular acidosis, may have hypercalciuria and develop renal stones. The term "Fanconi's syndrome" is sometimes used to describe the more complex tubular defects which in children are often due to cystinosis, and in adults may be associated with copper retention in Wilson's disease. Both these conditions are inborn errors of metabolism, but vitamin-D-resistant osteomalacia may also be seen following chronic pyelonephritis and uretero-colic anastomosis (Dickinson, 1960).

Any condition causing osteomalacia may be complicated by secondary hyperparathyroidism due to hyperplasia, or rarely adenoma, of the parathyroid glands (Davies *et al.*, 1956; Stanbury *et al.*, 1960). It is possible that hypocalcaemia always results in a compensatory increase in the secretion of parathyroid hormone.

I have recently been studying a small group of patients with osteomalacia. Only six out of the 16 patients discussed in this paper have been under my care, but eight patients in all were found to have osteomalacia while attending a department of physical medicine.

Three patients were undergoing treatment for steatorrhoea before they developed symptoms of bone disease (Table I), but osteomalacia was not diagnosed for two, three, and 18 years respectively. A further eight patients were found to have steatorrhoea when a three-day fat-balance study was carried

TABLE I

Diagnosis	Males	Females
Steatorrhoea:		
Coeliac disease		1
Idiopathic	1	1
Occult steatorrhoea:		
Idiopathic		2
? Hodgkin's disease		1
Partial gastrectomy	1	
Partial gastrectomy and horseshoe kidney		1
Partial gastrectomy and biliary cirrhosis	1	
Cirrhosis of liver		2
Polycystic kidneys		1
Spastic quadriplegia		1
Rheumatoid arthritis and ? polyarteritis		1
Patients over 70		2

out in hospital. Two of these patients were known to have cirrhosis of the liver with biliary obstruction, and three patients had undergone a partial gastrectomy (Polya). One of these patients was known to have a horseshoe kidney, chronic pyelonephritis, and renal stones before she had a partial gastrectomy for benign adenomatous polyps, and another patient developed biliary obstruction and cirrhosis of the liver following a partial gastrectomy for chronic duodenal ulcer.

One patient is suspected to have Hodgkin's disease. Enlarged glands in the neck were treated with radiotherapy ten years ago, and she has two discrete punched-out lesions visible on a lateral radiograph of the skull, suggestive of myelomatosis. There is no other evidence for this disease.

One patient was known to have polycystic kidneys, chronic pyelonephritis,

chronic renal failure, and acidosis for five years before she developed bone pain, but the diagnosis of osteomalacia was delayed for a further five years.

One patient was discovered to have osteomalacia when she was admitted to hospital seven years ago for investigation of a spastic quadriplegia. Despite considerable disability and deformity, she had never complained of bone pain until recently, when I discovered she had for some months stopped taking 2 mg. of calciferol daily.

One patient, a woman of 72 with chronic rheumatoid arthritis, had for several years complained bitterly of her rheumatism, though the arthritis appeared to be quiescent. She was for over three years known to have a severe and persistent anaemia, hypertension, and a number of small round opacities in the lung fields on chest radiography. Polyarteritis was suspected. Biochemical investigations supported a diagnosis of osteomalacia, and a daily dose of 200,000 i.u. of vitamin D appeared to relieve her symptoms. Steatorrhoea was suspected but not proven. Unfortunately she died shortly afterwards and a post-mortem examination was refused.

Two women, aged 79 and 73, were found to have osteomalacia without any evidence of steatorrhoea or renal disease. One patient had received radiotherapy for suspected myelomatosis, and the other fractured several ribs in a fall and subsequently developed pneumonia.

I have indicated the delay between the onset of symptoms and the diagnosis of osteomalacia in Table II. Few of these 16 patients developed symptoms

TABLE II

Duration of Symptoms in Years	Number of Cases	Duration of Symptoms in Years	Number of Cases
0	2	7	1
2	4	11	1
3	2	17	1
4	2	18	1
5	1		

suggestive of bone disease before the age of 45, and the majority were in the sixth decade (Table III). It is not surprising that their pains were attributed to spondylosis, to osteoarthritis, and even to "rheumatoid arthritis".

Patients suffering from osteomalacia may have signs and symptoms due to (1) weak and fragile bones, (2) hypocalcaemia, and (3) underlying diseases.

Skeletal deformity, and even fractures, may be painless, but patients

TABLE III

Age at Onset of Symptoms (Years)	Males	Females
0–10		
10–20		1
20–30		
30–40		
40–50	1	3
50–60	1	
60–70	1	7
70–80		2

commonly complain of skeletal pain. This is often a generalized ache, affecting muscles rather than joints, tending to be symmetrical and most marked in the back and lower limbs (Table IV). Backache is often dorsal, and pain tends to

TABLE IV

Symptoms	Number of Patients	Comment
Back pain	13	Radiating to groin or abdomen in 7
Pain in legs	13	
Shooting pain in groin	6	Stress fractures of femur and rami of pubis/ischium in 6
Painful knees	5	
Painful ankles	3	
Pain in foot	1	March fracture
Pain in arms	4	
Painful elbow	2	
Painful hands/wrist	2	

radiate to the front of the chest or abdomen. It may be very long-standing and is often intermittent, sometimes with pain-free intervals lasting months or years. Pain is usually aggravated by movement, especially after resting, but is absent at rest. It is often relieved, at any rate for a time, by analgesics such as aspirin or phenylbutazone, and by physiotherapy. Sometimes the pain appears to be fairly well localized to peripheral joints such as knee, ankle, wrist, or elbow, and a diagnosis of arthritis is made. One patient had a Judet arthroplasty for osteoarthritis of the hip in 1950, at the age of 49. Pain persisted, and she

later developed backache and jaundice. In 1957 osteomalacia was diagnosed, and the pain in her hip was relieved by calciferol within a few weeks.

Patients may notice tenderness of the bones, especially the lower ribs and spine, and are unable to turn over in bed without pain.

Acute pain, sudden in onset, following some trivial injury or occurring while walking, stooping, or even sneezing, is often due to a stress fracture of a rib, vertebral body, or the ramus of the pubis or ischium (Table IV). In one of my patients both clavicles fractured while she was being carried upstairs by the ambulance attendants. It was difficult to see the fractures, the bones were so porotic, and she was referred from a casualty department with a request for short-wave diathermy for a "strained neck". Fractures of the pelvic rami often cause a shooting pain in the groin on walking. Some do not appear to heal, and the bone ends are displaced. The patient may experience so severe a pain on movement, such as sitting up on a couch, that they cry out and are thought to be hysterical.

There may be few significant physical signs of bone disease on examination. Patients with congenital renal tubular defects, or steatorrhoea due to coeliac disease, may be short in stature. Most patients have a dorsal kyphosis, and less commonly a scoliosis. Recent shortening of the spine is evident if there are redundant skin folds on the abdomen, and if the distance measured between the crown and the pubis is less than that between the pubis and the heel. The spine is usually very stiff, and movements at the hip-joints may be very restricted, possibly because of muscle spasm, for a fuller range of passive movement can often be obtained if the patient's limbs are handled gently. Stiff hips are usually associated with fractures of the ramus of ischium or pubis, or of the femoral neck and shaft.

Patients may notice tenderness of the bones, and on examination the sternum, ribs, spinous processes, iliac crests, and tibiae, in particular, are often very tender, even on light percussion.

Tetany is uncommon, possibly because the parathyroid glands maintain a relatively high level of ionized calcium in the body fluids, but patients often complain of cramps and paraesthesiae. Dent and Garretts (1960) have recently described an uncommon but definite association between skin lesions and hypocalcaemia, and this possibly must be considered if patients do develop skin disease.

Muscular weakness is often severe, and may cause the patient to become bedridden for no apparent reason; or they may find that they have not the strength to lift a kettle or to climb stairs. Weakness of the hip muscles may be partly responsible for the characteristic "waddling gait", and for the difficulty which some patients experience in starting to walk when they first stand up

(Rose, 1956). I noticed that hypotonia tended to mask spasticity due to hemiplegia and paraplegia in two patients, and this became more of a handicap when their bone disease was treated.

Although steatorrhoea is commonly associated with osteomalacia in Britain, it may be symptomless. However, patients sometimes complain of weight loss, abdominal discomfort, and recurrent episodes of diarrhoea, and in severe cases the stools are pale, frothy, and bulky. There may be a history of residence in the tropics, of partial gastrectomy, of liver disease, or of previous episodes of rheumatism. Examination may reveal pigmentation of the skin and mucous membranes. Anaemia, soreness of the tongue, bruises, or oedema will suggest impaired absorption, not only of fat, but also of iron, vitamin B_{12}, folic acid, protein, and other vitamins including B complex and vitamin K. When steatorrhoea complicates biliary cirrhosis the liver is often enlarged, the skin and conjunctiva may be jaundiced, and the patient complains of nausea, vomiting, and pruritus.

Renal disease will be suspected if there is a history of recurrent urinary infections, thirst, polyuria, nocturia, or episodes of extreme muscle weakness. The kidneys may be palpable.

INVESTIGATIONS

Investigation of a patient with suspected osteomalacia will determine (1) whether bone disease is present or not; (2) whether there is a demonstrable disorder of calcium metabolism; and (3) whether some predisposing condition is present.

X rays reveal generalized rarefaction of the skeleton only when it has lost at least a quarter of its total calcium content, and this will take several years. Radiographs may therefore be normal even when the patient has bone pain and abnormal biochemistry (Dent and Hodson, 1954). When osteomalacia is gradual in onset the bones become rarefied and bend at points of stress. The clavicles droop, and the ribs are crowded together and slope more acutely downwards, so that the chest appears to be triangular in shape. The pelvis may become deformed. The vertebral bodies become more translucent, with prominent pencilled cortices. Ballooning of the intervertebral disk may make them biconcave, or the nucleus pulposus may herniate into the vertebral body. If the remaining trabeculae collapse, the vertebra becomes wedged or flattened. Gross changes are easily recognized, but they are not, of course, specific for osteomalacia, and similar changes may be seen in any generalized bone disease which causes rarefaction of the skeleton. It is sometimes difficult, in fact, to determine whether there are significant changes in the spine on X-ray examination.

Radiography of the chest and pelvis may reveal multiple, and sometimes unsuspected, fractures of the ribs and of the rami of pubis and ischium (Table V), and there may be distortion of the obturator foramen, due apparently to

TABLE V

Findings on X-ray Examination	Number of Patients
Rarefaction	16
Kyphosis	10
Scoliosis	3
Fractures:	
Rami of pubis/ischium	9
Ribs	6
Femora	4
Scapula	2
Vertebral body	1
Clavicles	1
Metatarsals	1
Metacarpals	1
? Tibia	1
Secondary hyperparathyroidism	1

displacement of bone fragments (Plate XXI, Fig. 3). Fractures may occur in other bones such as the femur or metatarsals (Plate XXI, Fig. 4), and small cortical stress fractures may be seen, especially along the shaft of tibia and femur. These must be distinguished from those which occasionally occur in Paget's disease, Cushing's syndrome, and polyostotic fibrous dysplasia (Albright and Reifenstein, 1948).

Certain characteristic changes are seen radiologically if osteomalacia is relatively sudden in onset (Dent and Hodson, 1954). The bones may appear surprisingly normal, but rarefied zones are seen extending across the shafts of the long bones, and at certain characteristic sites such as ribs, pelvic rami, lateral border of scapula, and neck of femur. These translucent zones consist of uncalcified osteoid (Milkman, 1934; Albright and Reifenstein, 1948). They may develop at the site of stress fractures, but it has been suggested that they are produced by the pulsation of overlying arteries (Le May and Blunt, 1949; Steinbach *et al.*, 1954). The evidence is not very convincing. In the pelvis these stress fractures and translucent zones seem to coincide with the junction between pubis and ischium. Patients with Milkman's syndrome may subsequently develop other radiological features of osteomalacia (Plate XXI, Fig. 5), and translucent zones may be associated with marked skeletal rarefaction.

If osteomalacia is complicated by secondary hyperparathyroidism, characteristic subperiosteal erosions may be seen radiologically, especially in the phalanges of the hand (Plate XXI, Fig. 6), but they may also be seen at the wrists, the upper end of the tibia and humerus, the femoral neck, and round the rami of the pubis and ischium; the skull may present a uniform granular appearance.

The most effective way of proving a diagnosis of osteomalacia is to demonstrate the presence of osteoid seams in bone removed at biopsy, but the technique and interpretation of sections are not easy, and bone biopsy is seldom performed.

I do not propose to discuss laboratory investigations in detail, but these usually reveal a low or normal serum calcium, a low plasma phosphorus, a raised serum alkaline phosphatase, and a reduced output of calcium in the urine. I would emphasize the fact that deviations from normal values may be slight, and, if osteomalacia is suspected, tests should be repeated on several occasions before the diagnosis is discarded. I have summarized the results of those tests which led to a diagnosis of osteomalacia in this group of 16 patients (Table VI). Some values for serum calcium, phosphorus, and alkaline phos-

TABLE VI

Serum Calcium (mg./100 ml.)	No. of Cases	Plasma Phosphorus (mg./100 ml.)	No. of Cases	Serum Alkaline Phosphatase (K.-A. units/100 ml.)	No. of Cases
6·5–7·0	1	1·0–1·5	1	Over 100	1
7·0–7·5	1	1·5–2·0	2	Over 90	1
7·5–8·0	2	2·0–2·5	5	40–50	3
8·0–8·5	4			30–40	2
8·5–9·0	5	2·5–3·0	2	20–30	3
		3·0–3·5	3	15–20	1
9·0–9·5	2	3·5–4·0	3		
9·5–10·0	1			Under 15	5

phatase were apparently within normal limits, but the results of the other tests were abnormal. It is evident that isolated estimations of calcium, phosphorus, or alkaline phosphatase can be misleading.

If steatorrhoea is gross, analysis of a random stool may show fat globules on microscopy, or a fat content which exceeds 25% of the dry weight. However, it is usually necessary to admit the patient to hospital and carry out a fat-balance study, preferably over a five-day period. Steatorrhoea can be due to pancreatic disease, but a flat sugar-tolerance curve and a low serum protein level suggest

defective absorption from the small intestine. The patient may have a normocytic, microcytic, or macrocytic anaemia, with a low serum iron and vitamin B_{12} level, and a megaloblastic bone-marrow. If diarrhoea is severe the patient can develop sodium and potassium deficiency. If chronic liver disease is present an elevated serum alkaline phosphatase may be rather misleading. An abnormal flocculation pattern in the small intestine may be seen following a barium meal.

Patients with primary renal disease may have chronic uraemia and acidosis. Examination of the urine may reveal albumin, sugar, or amino-acids, and an excessive excretion of calcium or phosphates. Straight radiographs of the abdomen may show nephrocalcinosis or renal stones, and an intravenous pyelogram may reveal abnormal or poorly excreting kidneys.

The patient suspected of having osteomalacia can be fully investigated only in hospital, but certain screening tests can be carried out initially in the outpatient department, and include: (1) Radiographs of the abbreviated skeleton: lateral view of skull, thoracic and lumbar spines; postero-anterior view of chest; antero-posterior view of hands, wrists, and knees for evidence of hyperparathyroidism, of pelvis including hip-joints, and of abdomen to show renal calcification. (2) Estimations of serum calcium, plasma phosphorus, serum alkaline phosphatase, blood urea, plasma proteins, alkali reserve; haemoglobin concentration, white cell count, mean cell haemoglobin concentration and mean cell volume, erythrocyte sedimentation rate. (3) Examination of urine for sediment, protein, sugar, *p*H, and calcium content (Sulkovitch test); 24-hour urinary calcium excretion. (4) Examination of a random stool for fat content and microscopy.

Osteomalacia and osteoporosis must be distinguished from other generalized bone and joint diseases, including ankylosing spondylitis, hyperparathyroidism, osteogenesis imperfecta, generalized carcinomatosis and multiple myelomatosis, and infiltration of bone in leukaemia, polycythaemia, and Hodgkin's disease.

CONCLUSION

It is evident that we know very little about metabolic bone disease. Despite a great deal of research during the last thirty years, we are still ignorant of the process by which osteoid is normally produced and calcified, and of the role of alkaline phosphatase, magnesium, and citrates. We do not know the exact function of parathormone, or even whether the parathyroid glands produce more than one hormone. We know little definite about the action of vitamin D, apart from the fact that it promotes absorption of calcium from the intestine, and less about the effect of hormones derived from the thyroid, adrenal cortex, and gonads.

X rays reveal only advanced bone disease, and biochemical investigations are often complex and require the patient's admission to specialized metabolic units. Random estimations of the blood levels of calcium, phosphorus, and alkaline phosphatase are often misleading, for disturbances of mineral metabolism are not necessarily accompanied by constant or significant changes in blood levels. In any case, the level of serum calcium, for example, gives no information about the absorption of calcium, its turnover in the bones, or its excretion from the body.

It has been suggested that osteoporosis may account for the fact that fracture of the neck of the femur is much commoner in women than men after the age of 50 (Cooke, 1955; Buhr and Cooke, 1959). Perhaps bone disease accounts for the destructive changes which are sometimes seen in osteoarthritic hips, and for pain which we usually attribute to osteoarthritis or to rheumatoid arthritis.

I do not suggest that osteomalacia is a common cause for skeletal pain, for I have been able to confirm this diagnosis in only a few patients during the last five years. But it must be suspected if a patient admits to symptoms suggestive of steatorrhoea, hepatic or renal disease, or gives a history of a partial gastrectomy. Patients with gross bone disease are still being treated with analgesics, prolonged physiotherapy, psychotherapy, and even surgery, when their symptoms could be completely relieved by taking a few milligrammes of vitamin D daily.

When confronted with an atypical joint condition I am often reminded by the well-known couplet, "When in doubt, think of gout", to have the serum uric acid estimated. I am tempted to coin another, though less elegant, phrase—"When patients moan, think of bone". Unfortunately the investigations are more complex, time-consuming, and expensive.

Acknowledgments

I am indebted to my colleagues at the Middlesex Hospital, and in particular to Dr. A. C. Boyle, Dr. G. D. Hadley, and Dr. J. D. N. Nabarro, for allowing me to study patients under their care.

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FIG. 1.—Radiograph of pelvis of patient bedridden with chronic rheumatoid arthritis.

FIG. 2.—Radiograph of lumbar spine of man aged 26 suffering from idiopathic osteoporosis. Note skeletal rarefaction, coarse vertical trabeculae, and crush fracture of second lumbar vertebra.

FIG. 3.—Radiograph of pelvis of woman aged 67 with osteomalacia, showing generalized rarefaction, stress fractures of rami of pubis and ischium with overlapping of bone fragments and distortion of obturator foramen. Note translucent zone in left pubic ramus.

FIG. 4.—Radiograph of feet of patient with osteomalacia and secondary hyperparathyroidism, showing march fracture of second right metatarsal.

FIG. 5.—Radiograph of pelvis of man aged 58, showing bilateral stress fractures of neck of femur and translucent zone in left pubic ramus.

FIG. 6.—Radiograph of hand showing subperiosteal erosions in phalanges characteristic of hyperparathyroidism.

PLATE XXII

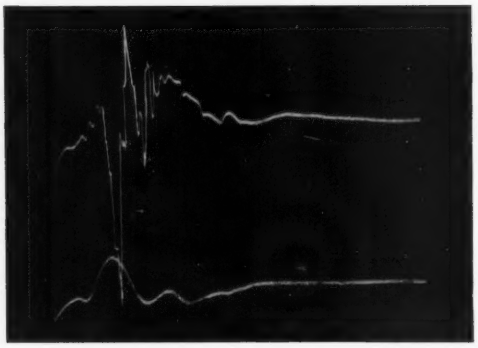

FIG. 1.—Tracings produced with (A) needle and (B) skin electrodes.

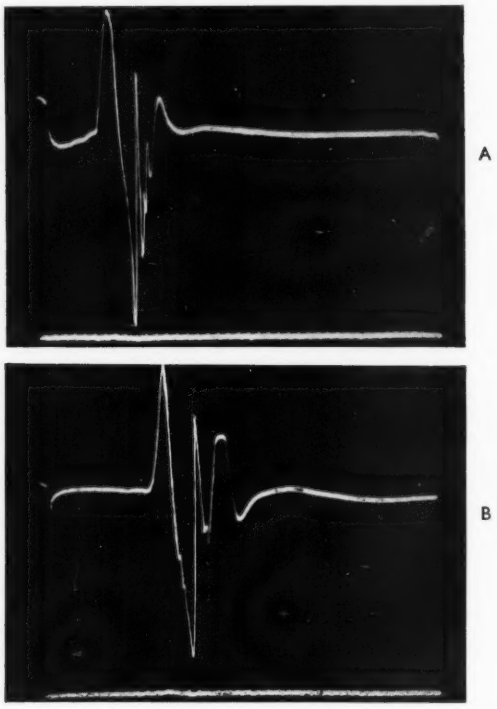

FIG. 2.—Tracings showing difference in latency on stimulation of ulnar nerve at (A) wrist and (B) elbow.

ORIGINAL PAPER

NORMAL VARIATION OF NERVE CONDUCTION IN THREE PERIPHERAL NERVES

By B. G. B. CHRISTIE and E. N. COOMES

From St. Thomas's Hospital, London

In recent years applied physiology has begun to play an important part in medical diagnosis. The object of this paper is to provide some base-line normal values for nerve conduction in three peripheral nerves—the ulnar, median, and lateral popliteal nerves—as these nerves are frequently the subject of conduction tests. It has been well described by Weiss (1956) that when a nerve is constricted there is generalized thinning of the axon distal to the point of constriction. Conduction velocity in nerve is proportional to the calibre of the axon (Hodes *et al.*, 1948), and this proportionality is valid also for nerves after injury (Hursh, 1939). It therefore follows that, as the calibre of the axon decreases, so also does conduction velocity.

Now that more modern techniques have been developed and concentric needle electrodes are standard equipment it is felt that a series of normal subjects should be examined, their conduction velocities and latencies recorded, and the artifacts encountered discussed. In previously published series skin electrodes (Hodes *et al.*, 1948; Norris *et al.*, 1953) and coaxial needle electrodes (Thomas *et al.*, 1959) have been used to pick up the action potential from the activated muscles.

MATERIALS AND METHODS

Conduction velocity was measured in thirty medical and physiotherapy students on three nerves—the median, ulnar, and lateral popliteal. The equipment used was the standard electromyograph at St. Thomas's Hospital, London, designed and developed in the electronic section. A bipolar electrode consisting of two saline-soaked tufts 1 cm. in diameter and 2 cm. apart was supplied from a balanced constant-voltage stimulator delivering a rectangular pulse of 0·1 millisecond duration percutaneously. A concentric needle electrode was used to pick up the action potential from the muscle under study, and this was photographed synchronously from the cathode-ray oscilloscope on the electromyograph.

To decrease skin resistance and current distortion at the source of stimulation the skin over the nerve under study was prepared with an abrasive paste consisting of pumice suspended in glycerin. This partially removed the stratum corneum of the epidermis, which layer offers the highest impedance to the

passage of current. Two points along the course of the nerve were stimulated and the recordings photographed. Measurements of distance were taken on the surface of the skin from the point of skin puncture to the mid-point of the bipolar stimulator over the nerve.

Three groups of calculations have been made: (1) the conduction velocity in metres per second, which was measured between the two points of stimulation, and the terminal velocity—that is, the velocity between the distal point of stimulation on the nerve and the recording electrode; (2) the latencies of the action potentials following stimulation of the nerve at the two levels were measured in milliseconds; and (3) the duration of the action potentials from the initial deflection of the phenomenon to its end was measured in milliseconds. To assess the accuracy of the method of measurement these readings have been repeated four times on one subject on different days (Table I).

TABLE I

VALUES OBTAINED ON THE SAME SUBJECT (MALE AGED 23) ON FOUR DIFFERENT DAYS

Nerve Tested	Conduction Velocity (metres per sec.)	Terminal Velocity (metres per sec.)	Duration (milli-seconds)	Latencies (milliseconds)	
				Wrist	Elbow
Ulnar nerve ..	49·0	22·0	15·0	4·5	10·0
	63·0	30·0	14·0	4·0	10·0
	58·0	27·8	14·0	4·0	9·0
	63·2	34·0	11·5	3·5	8·0
Mean	58·3	28·5	13·6	4·0	9·2
Median nerve ..	40·0	20·0	16·5	4·0	9·0
	58·2	15·0	15·5	4·0	10·0
	60·0	23·0	12·0	5·0	9·0
	58·0	17·5	14·0	3·0	8·0
Mean	54·0	18·9	14·5	4·0	9·0

All the investigations were carried out at room temperature, the subjects having been told to rest for fifteen minutes beforehand. No effort was made to heat up the limbs before testing, as these normal values were required for clinical work when elaborate heating precautions were not normally undertaken on patients. Examination of Table I shows that under these circumstances considerable variations may occur in the same patient from day to day. Tem-

perature variation in different muscles is quite marked (Buchthal *et al.*, 1944), and we are indebted to Professor Buchthal for pointing out that some of the variations may be due to temperature difference. Hendriksen (1956) found a mean alteration in conduction velocity of 2·4 metres per second (m.p.s.) per degree centigrade change in temperature (range 29°–38° C.).

RESULTS

Table II shows the velocity of conduction obtained by this method. Several points immediately become evident. First, the mean velocities agree fairly well with those recorded by other observers: Norris *et al.* (1953), who calculated the

TABLE II

VELOCITY OF CONDUCTION IN METRES PER SECOND

Median Nerve		Ulnar Nerve		Lateral Popliteal Nerve	
Conduction Velocity	Terminal Velocity	Conduction Velocity	Terminal Velocity	Conduction Velocity	Terminal Velocity
52·0	20·30	56·7	25·8	47·1	14·29
S.D. 8·6	S.D. 4·42	S.D. 10·8	S.D. 6·8	S.D. 10·0	S.D. 2·90

mean maximum velocity in the ulnar nerve as 58·1 m.p.s. in 25 subjects; and Thomas *et al.* (1959), who found a velocity of 56·2 m.p.s. to abductor digiti minimi and 55 m.p.s. to the first dorsal interosseus. The figures in the present series (Table I), however, show a wider spread of normal values, the standard deviation (S.D.) being as high as 10·8 m.p.s. Similarly, with other readings taken on the median and lateral popliteal nerves the standard deviation is still in excess of what would be expected. Whether this represents a true scatter of nerve velocity among the subjects under test is discussed later.

Table III shows the values obtained from measurement of the latency—

TABLE III

LATENCY MEASUREMENTS IN MILLISECONDS

Median Nerve		Ulnar Nerve		Lateral Popliteal Nerve	
Wrist	Elbow	Wrist	Elbow	Ankle	Knee
3·9	8·9	4·3	10·2	6·0	16·2
S.D. 0·3	S.D. 1·2	S.D. 0·5	S.D. 1·4	S.D. 0·6	S.D. 2·3

that is, the time taken for the stimulus to excite the muscle—from various standard points along the nerve. In spite of variations in length of the limbs, these results appear to be more constant, the standard deviations lying roughly between 7% and 14% of the mean.

TABLE IV

DURATION OF ACTION POTENTIALS IN MILLISECONDS

Median Nerve		Ulnar Nerve		Lateral Popliteal Nerve	
Wrist	Elbow	Wrist	Elbow	Ankle	Knee
15·7	15·9	14·00	14·1	14·9	15·3
S.D. 2·4	S.D. 2·6	S.D. 2·14	S.D. 2·0	S.D. 3·3	S.D. 2·9

Table IV shows the duration of the action potentials in various muscles as read from the photographic recordings. These figures are fairly constant in the neighbourhood of 15 milliseconds with a standard deviation of about 2·5 milliseconds in all the nerves examined (N = 30). This set of readings is of importance for reasons that are discussed later.

DISCUSSION

The value of nerve conduction velocity measurements as such as an electrodiagnostic aid is rather suspect. Figures that have been produced show a large scatter of values. Whether this represents a true scatter of velocity amongst normal subjects is a point for debate; for example, in the case of the lateral popliteal nerve figures of 27 m.p.s. and 67 m.p.s. would lie in the normal expected range—the normal expected range being the mean plus or minus twice the standard deviation. This seems unlikely.

On looking at the diagram several points of error become evident. The measurement of the length of the nerve under question is made on the surface of the skin, but the actual point of "firing off" of the nerve impulse on stimulation is a matter for conjecture rather than of exact measurement. In this investigation the mid-point between the bipolar stimulators was taken as the point of origin of the nerve volley. However, on looking at the diagram it can be seen that there is no means of knowing where the maximum concentration of current lies; points *a*, *b*, *c*, or a^1, b^1, c^1, could all equally well be the origin of the nerve volley, and in this case there may be several centimetres difference from the value measured on the skin. As shown in the Figure, this measurement must only be an approximation for the length of the nerve. Therefore in the measurement of the velocity of conduction along the trunk of a nerve this

uncertainty alone could account for an error of as much as 15%. Similarly, when considering the "terminal velocity" in a nerve—that is, the "mean" velocity between the distal point of stimulation and the needle electrode—a similar error would be an even more important factor. But this factor cannot account for the extreme slowing that apparently occurs in nerve propagation. This can, however, be accounted for by considering the diagram again. When the latency is measured, this must include the time taken for the propagation of the action potential along the nerve fibre for transmission at the neuromuscular junction, and for the propagation of the action potential along the muscle fibre itself to the site of the needle electrode.

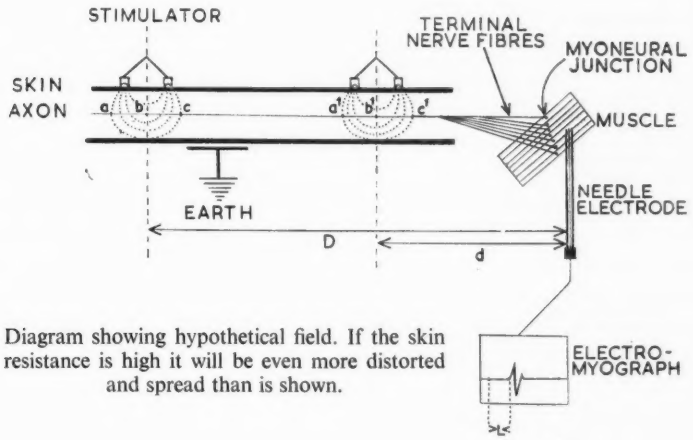

Diagram showing hypothetical field. If the skin resistance is high it will be even more distorted and spread than is shown.

It is known that there is thinning of the axon as it passes distally, and that the most terminal nerve fibres are considerably thinner than those found proximally. This could account for some slowing, as nerve velocity is proportional to the calibre of the nerve fibre; but in our figures the "mean terminal velocity" was found to be only about one-third of the reading in the main trunk, and it is not considered that this decrease in calibre is the sole factor.

Neuromuscular transmission delay has never been accurately measured, but estimates have been made, rather arbitrarily, of the order of 0·5 millisecond. It is almost impossible to measure this directly, but it is reasonable to suppose that the time taken for this to occur is of the same order as that for synaptic conduction in other parts of the nervous system, which has been estimated accurately (Eccles, 1957).

The propagation of action potential along muscle fibres has been measured directly by Buller *et al.* (1959) as 3–4 m.p.s. and by Buchthal *et al.* (1955) as

4·0 m.p.s. S.D. 0·5 m./sec., N = 16, 35·5° C. If, then, the needle electrode was 1 cm. away from the motor point of the muscle under study a latency artifact of the order of 3 milliseconds would be produced. When dealing with latencies of the order of 6 milliseconds this artifact becomes of proportions formidable enough to account for an apparent reduction of 50% in velocity as measured by the technique described.

If one bears in mind this additional error, measurement of the latencies (Table III) alone is probably sufficient. This confirms the work of Simpson (1956), who found that in the diagnosis of median-nerve compression by electrodiagnostic means latency measurement sufficed. In his controls there was no great deviation, the normal range being 3–4 milliseconds for the latency of median-nerve conduction at the wrist.

From examination of our figures for latency it becomes evident that the normal scatter is of the order of 10–20% from the mean, which is much lower than the values obtained on attempted conduction-velocity measurements. Variation in size of the limbs under study must play some part, but the variations produced on account of this are much less than those observed when conduction velocity is measured. In support of measurement of latencies, the values obtained from measurement of conduction velocity made on the same subject on different days show that the nerve velocity measurements apparently vary from day to day, probably owing to temperature variations, and the spread of values for velocity in the same subject is in the order of 10–15%. The values for the latencies, however, remain more constant.

It is important to know the duration of the normal action potentials as shown in Table IV. Bauwens (1958) has emphasized that not only does the velocity of conduction of the fastest nerve fibres decrease when a nerve is compressed, but there is often a scatter and breakdown of the normally well-integrated action potential. This has been interpreted by him as differential nerve velocities distal to the point of constriction. Bearing this in mind, therefore, it is important to know what would be the normal expected range. The figures obtained from thirty normal subjects are fairly consistent—around 15 milliseconds duration with approximately a standard deviation of 2–3 milliseconds. Cöers and Woolf (1959) stated that for certain muscles the end-plates and terminal nerve-endings all lie in a band about the middle of the muscle belly, the mid-point of the muscle fibre. This does not hold for all muscles, the exceptions being the gracilis and sartorius muscles. The small scatter of our figures, considering the different distances of the needle electrode from the neuro-terminal apparatus, would tend to support Cöers and Woolf's statement of localization of end-plates.

The relative merits of needle electrodes and surface electrodes are not

really within the scope of this paper. For straightforward measurement of nerve propagation, no doubt surface electrodes are quite as good as needle electrodes (Plate XXII, Fig. 1), but when considering breakdown or temporal dispersion of the muscle action potential following nerve stimulation (Plate XXII, Fig. 2) it is doubtful whether such an accurate picture would be reproduced with surface electrodes, and it is in the abnormal cases that this technique is used.

CONCLUSIONS

1. For routine electrodiagnostic investigation of nerve conduction the measurement of latency is probably all that is required. Measurement of the apparent length of the nerve on the skin surface increases the error, probably by up to 25%, and brings in unnecessary complications.

2. The duration of the muscle action potential appears to be fairly constant, with no more variation than would be expected from differing needle positions in the muscle in relation to the end-plates.

3. For physiological investigations of normal subjects surface electrodes are quite sufficient, but for the investigation of the abnormal case needle electrodes are superior.

SUMMARY

Thirty young subjects had their velocity of conduction, latency of nerve conduction, and duration of muscle action potential measured in the ulnar, median, and lateral popliteal nerves. The results are recorded and their relative values discussed and assessed.

ACKNOWLEDGMENTS

The authors acknowledge their gratitude to Dr. Philippe Bauwens, of the Physical Medicine Department, St. Thomas's Hospital, for his encouragement during the course of this work. They are also indebted to Professor Fritz Buchthal, of the Institute of Neurophysiology, University of Copenhagen, who kindly read the manuscript and offered much help and criticism.

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[References concluded at foot of page 326

ORIGINAL PAPER

NEUROTICISM IN UNSELECTED OUT-PATIENTS ATTENDING PHYSICAL MEDICINE AND ORTHOPAEDIC DEPARTMENTS*

By PETER SAINSBURY

M.R.C. Clinical Psychiatry Research Unit, Graylingwell Hospital, Chichester

My excuse for intruding into physical medicine is that, as a psychiatrist, I have been interested in the physiological accompaniments of emotional disturbance, especially the neuromuscular ones, and their relation to symptoms, such as backache or other muscular pains. Consequently, I especially wanted to include the departments of physical medicine and of orthopaedics in a survey of neuroticism in out-patient clinics which the Medical Research Council's Clinical Psychiatry Research Unit was undertaking. An opportunity to do this was very generously provided by Dr. Donald Wilson, the Director of Physical Medicine at the Royal West Sussex Hospital.

PURPOSE OF THE SURVEY

If our findings in the physical medicine clinic are to carry any conviction, it will be necessary first to outline the survey of which they form a part. Its purpose was to examine the often-doubted notion that psychosomatic diseases are a distinct and valid entity. In short, have the so-called psychosomatic illnesses any claims to be considered separately from other illnesses? Our method of doing this was to find whether out-patients with diagnoses commonly accepted as "psychosomatic" could be distinguished psychologically from those who have other diseases. The psychological criteria on which we chose to differentiate them were two traits, or dimensions, of personality: neuroticism and extraversion. These were selected because they were both hypothetically appropriate and because they can be reliably measured by a short questionnaire, the Maudsley Personality Inventory (Eysenck, 1959). An important and essential feature of this questionnaire is its validity—that is, it has been found consistently to differentiate a group of patients known to be neurotic from one which is not.

Neuroticism is defined as the general emotional lability of a person, his emotional over-responsiveness, and his liability to neurotic breakdown under stress. *Extraversion* refers to the outgoing, uninhibited, sociable proclivities of a person (Eysenck, 1959).

* Paper delivered at the Annual Meeting of the British Association of Physical Medicine, London, April 30, 1960.

Our hypotheses, therefore, were: (1) that patients with psychosomatic diseases would have a higher neuroticism score than controls; and (2) that they would score lower than the controls on extraversion. The theoretical reasons for these suppositions have been discussed elsewhere (Sainsbury, 1960).

METHODS

Through the kind co-operation of the consultants at the Royal West Sussex and St. Richard's Hospitals, Chichester, we were able for two months to ask all out-patients aged 20–59 to complete the questionnaire while they were waiting to see the doctor. A proportion of the patients escaped us, but these were later traced and found not to differ in any relevant respects from those seen. We therefore believed we examined a representative sample of out-patients, unselected except for age.

The patients' diagnoses were obtained and divided into four operationally defined groups: (1) a *psychosomatic* group: those diseases designated psychosomatic in at least two of six recent textbooks on the subject; (2) a *possibly*

TABLE I

DIAGNOSES WHICH OCCURRED EIGHT OR MORE TIMES

Psychosomatic	Possibly Psychosomatic	Controls
Asthma	Cervical erosion	Appendicitis
Coronary disease	Chalazion	Cholecystitis
Diabetes	Herniated disk	Deafness
Hypertension	Low-back pain	Ear infections
Peptic ulcer	Lupus erythematosus	Fractures
Pulmonary tuberculosis	Obesity	Haemorrhoids
Rheumatoid arthritis	Pain (no organic basis found)	Hernia
Thyrotoxicosis	Sterility	Injuries
Chronic nasal infection		Osteoarthritis
Dysmenorrhoea		Pregnancy
Irregular menstruation		Prolapse of uterus
Leucorrhoea		Skin infections
Menorrhagia		Tumours, benign
Acne vulgaris		Varicose veins
Alopecia		Warts
Dermatitis		Carcinoma
Eczema		Poliomyelitis
Fungus infections with hyperidrosis		Rheumatic heart disease
Pruritus		
Psoriasis		
Seborrhoeic dermatitis		
Urticaria		

psychosomatic group: diseases which some authorities claim may have a psychological cause; (3) a *neurotic* group: diagnoses made at the psychiatric clinic; and (4) a *control* group: all the remaining diagnoses (see Table I).

RESULTS

About 1,350 patients from 11 clinics completed the questionnaire. The clinics with the greatest proportion of patients scoring high on neuroticism were the general medical, skin, gynaecological, and physical medicine clinics, in that order (Table II).

TABLE II
CLINICS AND NEUROTICISM

Clinics	Above Median Score	
	No.	%
Psychiatric	99	77·3
Medical (including diabetic and chest)	109	51·9
Dermatological	109	49·6
Gynaecological	79	46·8
Physical medicine	75	43·4
Orthopaedic	74	43·5
Surgical	63	42·3
Ear, nose, and throat	30	38·5
Ophthalmic	19	33·3

Median neuroticism score of total sample = 22·64.

Next, it was found that the patients in both the psychosomatic and possibly psychosomatic groups had very significantly higher scores on neuroticism than

TABLE III
MEAN NEUROTICISM SCORES AND STANDARD DEVIATIONS OF THE FOUR DIAGNOSTIC GROUPS (AND OF THE GENERAL POPULATION)

Group		Neuroticism	N
Controls	546	18·38	10·79
Psychosomatic	459	23·81*	10·32
Possibly psychosomatic	231	23·10*	10·94
Neurotic	116	31·95*	9·15
Control quota sample (Eysenck, 1959)	1,800	19·89	11·02

* Very significantly different from controls.

had the controls, though they scored lower than the neurotics did (Table III). Similarly, the psychosomatic group were also very significantly less extraverted than the controls.

The finding that patients with psychosomatic diseases were more neurotic and less extraverted than those without (i.e. the controls) was confirmed when the diagnoses were separately examined (Table IV). Twenty-two psychosomatic

TABLE IV

NEUROTICISM AND EXTRAVERSION MEANS OF PSYCHOSOMATIC DIAGNOSES WHICH OCCURRED EIGHT OR MORE TIMES

Diagnosis	No.	Neuroticism		Extraversion	
		Compared with Controls	p	Compared with Controls	p
Dysmenorrhoea	9	Above	0·001	Below	NS
Urticaria	10	,,	0·01	,,	0·01
Leucorrhoea	13	,,	0·005	,,	NS
Fungus infections	9	,,	0·005	,,	NS
Alopecia	17	,,	0·005	,,	NS
Asthma	14	,,	0·01	,,	0·05
Eczema	32	,,	0·005	,,	NS
Menorrhagia	32	,,	0·005	,,	0·05
Seborrhoeic dermatitis ..	23	,,	0·01	,,	0·025
Psoriasis	28	,,	0·005	,,	0·025
Hypertension	19	,,	0·005	,,	0·05
Diabetes	37	,,	0·005	,,	0·025
Pulmonary Tb	54	,,	0·005	,,	0·01
Irregular menstruation ..	12	,,	0·001	,,	NS
Dermatitis	17	,,	0·005	,,	NS
Chronic nasal infection ..	14	,,	0·025	,,	NS
Thyrotoxicosis	14	,,	0·05	,,	NS
Rheumatoid arthritis ..	42	,,	0·05	,,	NS
Pruritus	13	,,	NS	,,	NS
Peptic ulcer	22	,,	NS	,,	NS
Acne vulgaris	9	,,	NS	,,	NS
Coronary disease	12	Below	NS	Above	NS
Control patients	546		18·38		25·74

NS = not significant.

diagnoses occurred sufficiently often to enable us to test whether the patients differed significantly from the controls on neuroticism. In 18 diagnoses out of the 22 the patients were, in fact, significantly more neurotic, and in only one of them, coronary disease, were they less neurotic. Of particular interest among

those high on neuroticism were the gynaecological diagnoses: menorrhagia, dysmenorrhoea, and leucorrhoea; the skin diseases: alopecia, eczema, seborrhoeic dermatitis, and psoriasis; the general medical diseases: diabetes, hypertension, and pulmonary tuberculosis, and, from the physical medicine department, rheumatoid arthritis, to which I will return later.

Patients with seven of the eight diagnoses in the "possibly psychosomatic" group—that is, the diagnoses for which some investigators claim a psychological cause—were more neurotic than the controls (Table V). Among those who

TABLE V

NEUROTICISM AND EXTRAVERSION MEANS OF "POSSIBLY PSYCHOSOMATIC" DIAGNOSES WHICH OCCURRED EIGHT OR MORE TIMES

Diagnosis	No.	Neuroticism		Extraversion	
		Compared with Controls	p	Compared with Controls	p
Low-back pain	43	Above	0·01	Below	NS
Prolapsed disk	37	,,	0·05	Above	NS
Lupus erythematosus ..	10	,,	NS	,,	NS
Sterility	8	,,	0·01	,,	NS
Cervical erosion	15	,,	0·01	,,	NS
Obesity	9	,,	NS	,,	NS
Pain (N.A.D.)	30	,,	NS	,,	NS
Chalazion	12	Below	NS	,,	NS
Control patients	546		18·4		25·7

were significantly more neurotic were patients with low-back pain and patients diagnosed as having a prolapsed intervertebral disk. It should be pointed out, incidentally, that this was the diagnosis made at the first interview—that is, when it was clinically probable, but before X-ray or other special investigation had confirmed the case as a typical one.

In the control list, on the other hand, there was no consistent relation between neuroticism or extraversion and diagnosis (Table VI). Patients with osteoarthritis, poliomyelitis, and fractures, for example, have normal scores on neuroticism. The fracture patients, however, were remarkable in another respect: they were unusually extraverted. In only two conditions of the 48 separately listed were the patients significantly extraverted. The liability to fracture therefore seems to be associated with more than normally outgoing and uninhibited tendencies in the personality, an observation not without relevance to the concept of accident-proneness.

TABLE VI

NEUROTICISM AND EXTRAVERSION MEANS OF CONTROL DIAGNOSES WHICH OCCURRED EIGHT OR MORE TIMES

Diagnosis	No.	Neuroticism		Extraversion	
		Compared with Controls	p	Compared with Controls	p
Warts	8	Above	0·01	Above	0·01
Prolapse	25	„	0·05	Below	0·01
Haemorrhoids	9	„	NS	Above	NS
Cholecystitis	8	„	NS	Below	NS
Ear infections	20	„	NS	Above	NS
Pregnancy	13	„	NS	„	NS
Osteoarthritis	25	„	NS	Below	NS
Appendicitis	12	„	NS	„	NS
Injuries	60	„	NS	Above	NS
Hernia	19	„	NS	Below	NS
Fractures	51	Below	NS	Above	0·05
Deafness	8	„	NS	„	(0·01)
Varicose veins	20	„	NS	„	NS
Tumours, benign	22	„	NS	Below	NS
Skin infections	14	„	NS	Above	NS
Carcinoma	16	Above	NS	Below	NS
Rheumatic heart disease ..	10	„	NS	„	NS
Poliomyelitis	9	Below	NS	Above	NS
Control patients	546		18·4		25·7

Before considering the physical medicine findings in more detail I would like to add two comments on the results described so far. The main conclusion —namely, that patients with psychosomatic diseases are more neurotic than those without—might have arisen because psychosomatic disorders tend to be chronic illnesses and consequently engender a secondary neurotic reaction. In order to exclude this explanation (Table VII) the neuroticism score of all those patients with chronic and seriously disabling illnesses—congenital deformities and poliomyelitis, for example—were compared with that of the controls. The neuroticism scores did not differ. It was concluded, therefore, that neuroticism is not dependent upon the chronicity of an illness. Secondly, the high neuroticism scores in psychosomatic cases may have arisen because hospital out-patient departments select neurotic patients, especially among those who are referred with the less serious conditions. The mean neuroticism score of the controls, however, was lower than that found by Eysenck for the normal popu-

TABLE VII

MEAN NEUROTICISM SCORE AND STANDARD DEVIATIONS OF PATIENTS WITH CHRONIC AND SERIOUS DISEASES, CONTROLS AND PSYCHOSOMATIC DISEASES

	Chronic or Serious Diseases	Controls	Psychosomatic Diseases
Number	67	546	459
Mean N	18·96	18·38	23·81
S.D.	9·08	10·79	10·32

lation of Great Britain; moreover, control diseases of a minor nature, such as skin infections, for which the more anxious patient may seek a specialist opinion, had low neuroticism scores. Thirdly, the results obtained may be ascribed to errors of sampling; but the patients who were omitted or had refused, when traced, were not found to differ by age, sex, or diagnostic category from those who completed the questionnaire.

BACKACHE AND RHEUMATOID ARTHRITIS

Mention has already been made of the relatively large proportion of patients scoring high on neuroticism in the physical medicine and orthopaedic departments, therefore it will be pertinent to consider the backache and rheumatoid arthritic cases in more detail.

Patients with a diagnosis of back strain or backache of uncertain origin were further studied in the following way. It was hypothesized that there were two groups of backache patients: those in whom physical causes and those in whom psychological causes were foremost. The cases were therefore divided, on the basis of their neuroticism scores, into the 15 who scored highest and the 15 who scored lowest. The case notes of the two groups were then compared on ten items of clinical information, to find whether the neuroticism scores also differentiated the groups clinically. It was found that in the high neuroticism group there was a significantly greater proportion of female patients, patients with a previous history of psychosomatic disease (commonly a gynaecological complaint), and those with a previous history of backache; patients with symptoms not related to the skeleto-muscular system and those with other muscular symptoms than backache approached, but did not quite reach, significance. The backache patients scoring high and those scoring low on neuroticism were not differentiated by age, by positive X-ray findings, by the presence of definite physical signs on examining the back, by straight leg raising, or by duration of the backache.

The patient's history and symptoms therefore had some characteristic relation to neuroticism, but the physical findings had not. This was not altogether unexpected, because in a previous experiment in which an electromyograph with a device for integrating the action potentials was used a significant correlation was found between a patient's rating on anxiety and the activity recorded in the forearm extensors and frontalis muscles. It was also found that muscular symptoms were common in these anxious patients, and that the electromyographic activity was higher in the area of the symptom than in the corresponding area in patients without the symptom (Sainsbury and Gibson, 1954). It was concluded from these observations that increased neuromuscular tension is a physiological accompaniment of anxiety, which when sustained may give rise to pain. Some evidence of a different kind for this view was obtained by analysing the replies to one of the items in the questionnaire used in the survey. The question was, "Would you rate yourself as a tense or highly strung individual?" It was answered positively by the backache patients significantly more frequently than by the controls; the same question did not, however, differentiate other psychosomatic patients from the controls.

The case notes of rheumatoid arthritics who scored highest and lowest on neuroticism were similarly examined. The 15 most and the 15 least neurotic patients were not differentiated on grounds of age, sex, severity of illness, or previous history of psychosomatic disorders. Of the nine patients studied, the only clinical aspect on which they differed was that those with a diagnosis of early rheumatoid arthritis were significantly more neurotic. These cases tended to be a little less clear-cut—in nearly half of them the Rose–Waaler test was negative, for instance. However, observations such as these based on retrospective examination of case notes can only suggest clues to be followed with caution.

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REVIEW SERIES

OSTEOARTHRITIS OF THE HIP-JOINT

By J. H. JACOBS

From the North Middlesex Hospital, London

THE problem of degenerative joint disease is becoming progressively more urgent as the expectation of life of the community rises and the number of people in the older age-groups increases. Since osteoarthritis is not a killing disease and methods of prevention are unknown, it has long been accepted as part of the normal process of ageing, and relatively little interest has been taken in its pathology and management compared with the amount of disability it causes. The search for improved surgical techniques has more recently stimulated inquiry into the natural history of the development of degenerative joint disease, and the papers written by Harrison, Schajowicz, and Trueta (1953) and by Lloyd Roberts (1953) have added considerably to our knowledge.

AETIOLOGY AND PATHOLOGY

Osteoarthritis of the hip-joint is most frequently seen in the older age-groups, and may be limited to the hip alone or be associated with degenerative changes in one or two other joints. More rarely, especially in women, it may occur as part of a primary generalized arthritis of the type described by Kellgren and Moore (1952). Not infrequently, especially when these changes occur in younger patients, some aggravating or casual factor which has involved the joint in excessive or abnormal strain can be demonstrated, and this type of the disease is normally referred to as secondary osteoarthritis.

Plewes (1940), in a review of 242 cases of osteoarthritis of the hip-joint, showed that some aggravating factor was present in over one-half of his patients. He listed the following conditions in order of frequency: old injury, slipped epiphysis, protrusio acetabuli, coxa plana, septic arthritis, dislocation, coxa vara, and Paget's disease. More recently Lloyd Roberts (1955), in a series of 124 cases requiring operation, found that 58·8% were of unknown aetiology and in the other 41·2% some precipitating cause could be discovered. Both these series were seen in orthopaedic clinics, and it is unlikely that the proportion of secondary arthritis would be so high in patients seen in physical medicine clinics, where the two most common aggravating factors are obesity and abnormalities in the contralateral limb such as shortening from any cause.

Many factors have been incriminated regarding the aetiology of primary osteoarthritis. Arteriosclerosis has frequently been postulated as a factor, since

osteoarthritis occurs so commonly in the older age-groups. However, Trueta and Harrison (1953) were unable to demonstrate any decrease in the arterial supply within the femoral head in old age, and they did not consider that cartilage degeneration was preceded or accompanied by ischaemic conditions within the femoral head. It is accepted that the joint changes occurring in tabes, syringomyelia, and peripheral neuritis are due to increased wear and tear on the joint, secondary to muscular hypotonia and diminished pain and proprioceptive sensibility, and it has been suggested that such changes may play a part in the development of osteoarthritis in patients who do not suffer from any overt neurological condition. No real evidence is available to substantiate this theory, especially as pain is one of the outstanding symptoms of degenerative joint disease. It is not surprising that both endocrine and metabolic factors have been incriminated, as certain circulating hormones tend to diminish in later life; however, no definite evidence is available to substantiate such a relationship and, despite the many suggestions put forward, the aetiology of the primary type of arthritis remains unknown.

Gowers coined the term "abiotrophy" to explain certain conditions of premature degeneration in the central nervous system, and it is not unreasonable to assume that certain individuals have a poorer-quality tissue than others, which tends to degenerate prematurely before completion of its allotted span of life. More recently Kellgren and Lawrence (1958), in a random survey of osteoarthrosis and disk degeneration, showed that the pattern of joint involvement and the severity of these changes were more pronounced, and the number of joints affected greater, in females than in males. They suggested that a major factor in the causation of osteoarthritis might be a sex-linked metabolic characteristic. Many of the structural changes so frequently found in aged, pain-free joints are very similar to those present in the clinically osteoarthritic joint; also it is not infrequent to find radiological osteoarthritic changes in a patient with a pain-free joint. At times trauma and excessive use may transform an aged, pain-free joint into a clinically osteoarthritic one, and this may possibly be due to extra stretching or tearing of the capsule with the subsequent onset of symptoms; but on the whole we can only speculate on the relationship between what appear to be two seemingly similar conditions.

A knowledge of the pathology and natural history of the osteoarthritic process occurring in the hip-joint is not purely of academic interest, as it can be shown that the development and advancement of these changes can be correlated with the progress of the clinical picture, and can also give a lead to the management of the disease and the application of rational physical treatment.

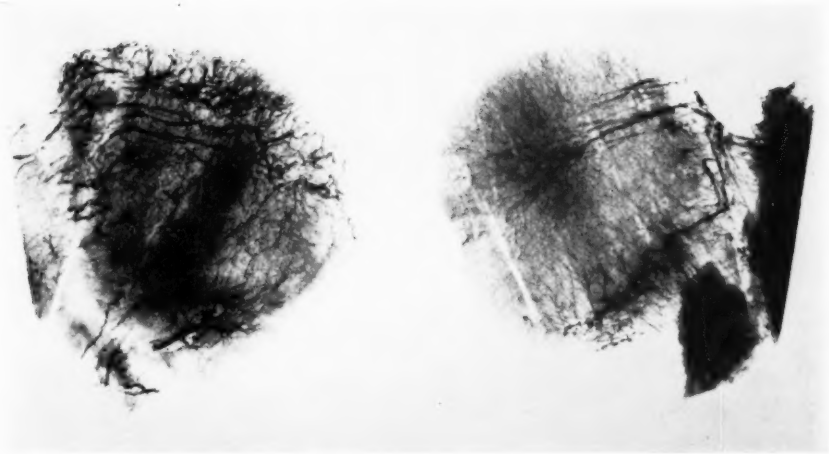
Pain in osteoarthritis may possibly arise in the cartilage and underlying

bone, capsule, and synovial lining, the surrounding muscles, or in the periosteum investing the intracapsular bone. If a joint is opened under local anaesthesia and the cartilage probed with a needle no pain is experienced, but on deeper probing of the bone ill-defined discomfort is felt, probably mediated via the intraosseous blood-vessels, which are supplied by nerve-fibres believed to be in part vasosensory. Probing of the synovial membrane also causes little discomfort, but on stretching the joint capsule intense pain is felt, and it seems probable that most of the symptoms experienced in osteoarthritis are secondary to changes occurring in the capsule.

It has been suggested by Lloyd Roberts (1955) that the capsular changes in osteoarthritis occur secondarily to the degeneration in cartilage and bone. It is therefore more rational first to describe the pathological changes occurring in the cartilage and bone, and then to see the effects these changes produce on the capsule and try to correlate them with the development and progress of the clinical picture.

Consideration of the anatomy of the hip-joint shows that the head of the femur, with the exception of the fovea capitis, is covered by hyaline cartilage, but that the acetabulum is only incompletely lined with a horseshoe-shaped area of cartilage. This arrangement is such that a considerable area on the infero-medial surface of the femoral head is prevented by lack of contact from transmitting joint stresses. Also it can be shown by joint section that anteriorly, superiorly, and to a less extent posteriorly, the marginal articular cartilage of the femoral head tends to project outside the acetabulum. The area of cartilage on the femoral head which articulates with the acetabulum has been termed the pressure area, and this area can be seen in slab radiography as the zone overlying the fan-shaped trabeculae which have developed as the result of pressure systems within the femoral head.

The earliest pathological change in osteoarthritis occurs in the articular cartilage, which loses its normal shiny bluish-white colour and becomes dull and yellow in appearance. At the same time a process of fibrillation or fissuring occurs on the surface of the cartilage, with the ultimate development of ulceration and exposure of the subchondral bone. Contrary to expectation, Harrison *et al.* (1953) have shown that in 71% of the femoral heads they examined degeneration was limited to the cartilage of the non-pressure areas, while in only 3% were these changes solely limited to the pressure zone. They explained these findings by postulating that, if excess of joint pressure was harmful to the healthy integrity of cartilage, then also lack of pressure was even more deleterious, possibly because the metabolism of cartilage was in part maintained by the mechanical pumping of synovial fluid by alternate compression of the cartilage, and that either excess pressure or lack of pressure prevented this



a *b*

FIG. 1.—Angiograms in the antero-posterior plane of the femoral heads of a subject aged 76. (*a*) The site of advanced osteoarthritis; (*b*) shows an excellent state of preservation. The arteries within the osteoarthritic head are dilated and increased in number, especially the epiphysial vessels.

FIG. 2.—Slab radiograph of an osteoarthritic femoral head, showing a large cyst beneath the pressure area communicating with the joint cavity by a small stoma.

FIG. 3.—Photomicrograph of part of the pressure area, illustrating how the bone-marrow differentiates into fibrocartilage.

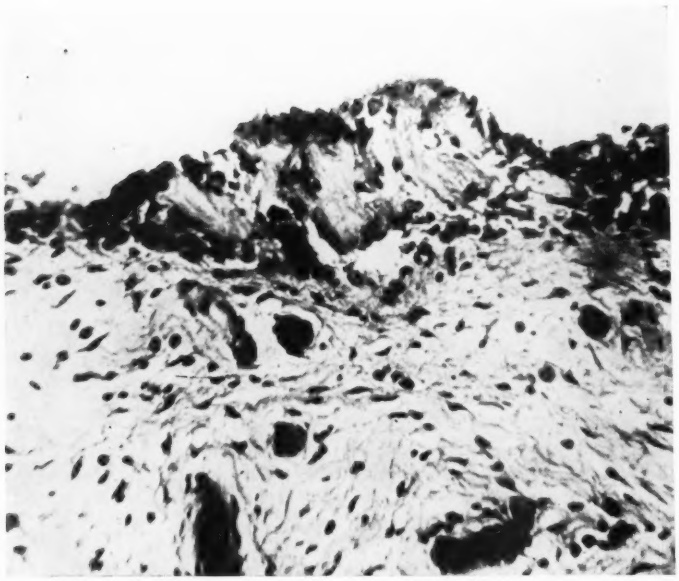


FIG. 1.—A group of cartilage fragments lying beneath the synovial surface.

FIG. 2.—Large fragment of cartilage lying below the synovial surface.

mechanical action. Thus both overcompression and lack of use might possibly lead to the same result. Subsequent to the degeneration and reduction in depth of the articular cartilage, the underlying subchondral calcified zone increases in thickness, and as the process continues the subchondral blood-vessels proliferate and enter the calcified zone. With the advance of the blood-vessels towards the joint surface, the areas where there was formerly normal articular cartilage become converted into regions consisting of highly vascular marrow, calcified cartilage, and bone covered by a layer of partially degenerate fibrocartilage.

This spread of vascularization together with the formation of overlying fibrocartilage and bone gives rise to the development of the osteophyte, and in the femoral head this process occurs earliest at the junction of the articular cartilage and synovial membrane—that is, at the margin of the fovea capitis medially and at the periphery of the articular cartilage laterally. The spread of osteophyte formation appears to be strictly limited to those areas within the joint which do not bear weight, and this tendency of osteophytes to grow into the free spaces outside the weight-bearing area allows maximal growth inferiorly. In advanced osteoarthritis these large inferior osteophytes may at times coalesce with those of the medial non-pressure area, and by their size cause a mechanical block within the joint.

In addition to the increase in small blood-vessels adjacent to the degenerate cartilage Harrison *et al.*, by means of their technique of injection and slab radiography, have shown that there is an increase in the large arteries within and around the osteoarthritic femoral head, and that this hypervascularity increases as the arthritic process advances (Plate XXIII, Fig. 1). This demonstration disposes of the theory that osteoarthritis occurs secondarily to ischaemic processes within the bone. Further evidence of this hyperaemic state can be drawn from the fact that the intra-articular temperatures in osteoarthritic joints are found to be raised above normal values (Fletcher *et al.*, 1954).

Following on the stage of hypervascularity produced secondarily to the degeneration of the cartilage Harrison describes a destructive phase within the femoral head. As the range of movement of the joint decreases, the body weight is transmitted through a progressively smaller area of the femoral head and the load per unit area is thus increased. The trabeculae of the head, which are already rarefied and weakened by the vascularity, tend to collapse, and the femoral head becomes distorted and begins to decrease in height from successive trabecular fractures.

Bone cysts are commonly present within the femoral head in advanced osteoarthritis, and Landells (1953) considers that these cysts invariably communicate with the joint cavity (Plate XXIII, Fig. 2) and are the result of intrusion

of synovial fluid through the watertight layer between the joint space and the cancellous bone, and that enlargement of these cysts is brought about by the slow persistent pressure of the synovial fluid on the weakened internal trabeculae.

Harrison points out that under certain circumstances, such as when mechanical conditions have been favourably changed, a reconstructive or partial healing process may occur in the femoral head by means of osteoclastic removal of necrotic bone, osteogenic activity within the pressure segment, and the appearance of fibrous marrow at the surface of the pressure area which later becomes converted into fibrocartilage (Plate XXIII, Fig. 3).

Lloyd Roberts has put forward the thesis that the changes in the synovial membrane and capsule found in osteoarthritis of the hip are largely due to the presence of articular debris, which he was able to demonstrate lying beneath the surface layer of the synovial membrane (Plate XXIV, Figs. 1 and 2). This debris arises from the degenerate joint surfaces and is removed by the synovial lining, resulting in congestion and a villous formation of the membrane. Following on this sustained synovial hyperplasia fibrosis occurs in the sub-synovial region and spreads to the underlying capsular areas. He considered that as a result of gravity most of the debris accumulated in the synovial pocket below the neck of the femur, and it was there that the greatest amount of synovial congestion occurred, so that it seemed probable that capsular fibrosis and shortening also began in that region and later spread to involve the entire joint capsule. This shortening and fibrosis of the capsule results in interference with the normal joint function, and an attempt can therefore be made to correlate the symptoms and signs found in osteoarthritis of the hip with the development of these capsular changes.

The position of greatest stability of the hip-joint during weight-bearing is one of extension with some abduction and medial rotation, and it has been shown that in this position the whole of the capsule is stretched and taut. Shortening of any part of the capsule will affect this position and result in painful stretching on attempted weight-bearing. As the fibrosis extends inferiorly, medial rotation and abduction as well as extension will also become restricted, and further shortening will cause deformity towards the position of flexion, adduction, and lateral rotation. In addition, reflex guarding by the muscles acting on the hip-joint occurs as a protective measure, and subsequently fibrotic changes also occur in these muscles, adding to the already deforming results brought about by the capsular fibrosis. These changes, by preventing the hip-joint from extending, limit the area of the femoral head which can come into contact with the acetabulum, and so hasten further deterioration in the already degenerating joint.

DIAGNOSIS

The cardinal symptoms of osteoarthritis of the hip are pain, stiffness, and diminution of activity. The pain is usually of slow onset and felt in the groin or buttock, but may at times be referred to the back of the knee-joint and give rise to the mistaken diagnosis of osteoarthritis of the knee. Occasionally the pain may be of sudden onset, and this may be due to a fracture of an osteophyte or impaction of a loose body, to haemorrhage or an effusion into the joint, or to stretching of the capsule or tearing of adhesions. The occurrence of osteoarthritic symptoms in a previously pain-free joint after trauma is well known and is frequently a matter of medico-legal interest, and can usually be explained on the basis of one of the above episodes. Pain, however, may sometimes be absent or tend to diminish considerably as the range of movement of the joint decreases, since the capsule and fibrotic muscles are gradually subjected to diminishing strain.

Early examination of the joint usually shows some loss of internal rotation, followed later by diminution of abduction and extension, and ultimately the hip may be held in flexion, adduction, and external rotation. With the development of fixed flexion of the hip-joint a lumbar lordosis occurs, and in the later stages, when the femoral head shows some degree of collapse and subluxation, a pelvic tilt may occur to compensate for the shortening of the affected hip, and these postural deformities may in their turn produce superadded back-strain. Wasting of the gluteal, quadriceps, and adductor muscles usually occurs as the disease progresses, and superadded adductor spasm may lead to increasing disability and deformity. When both hip-joints are affected, especially when flexion is limited, the patient may become severely disabled and find considerable difficulty in sitting down and in going to the toilet.

The X-ray appearances mirror the pathological changes already described. Loss of joint space indicates cartilage loss, sclerosis denotes eburnation and new bone formation in the area of calcified cartilage, and lipping of the articular margins and osteophyte formation are readily recognized. In the later stages collapse and deformity of the femoral head can be seen, and frequently bone cysts become visible, and these are invariably associated with advanced arthritis and pain.

The differential diagnosis should not give rise to great difficulty—intra-abdominal disease presenting with pain in the groin and a limp, a fracture of the femoral neck, especially in the elderly, and referred pain from the lumbar spine may at times be missed. Charcot's joints in the early phase may be painful; supertrochanteric bursitis, acromegalic joint disease, and para-articular calcification associated with paraplegia or prostatic sepsis may sometimes be misleading. "Shop-girl's hip", a postural strain due to prolonged standing with the

weight on one leg, may mimic the pains of osteoarthritis, while in secondary osteoarthritis it may be difficult to appreciate how much of the disability is due to the original disease and how much to the superadded degenerative changes.

TREATMENT

In the treatment of osteoarthritis of the hip one is at the disadvantage of dealing with a progressive lesion in a weight-bearing joint. This fact should be explained to the patient so that he can try and live within the limits set by his disability. The removal of any precipitating or aggravating factor such as obesity, or the correction of any mechanical defect such as shortening of a limb, can be a positive step in an attempt to prevent further joint deterioration, and an appreciation by the physician of the underlying pathology can help him to formulate a rational programme of physical treatment.

Physical Therapy

The principles of physical treatment apart from the correction of aggravating factors are: (1) to maintain the maximum range of movement in the hip-joint so that the body weight can be transmitted through as large an area of the pressure zone as possible; (2) to prevent capsular shortening and contractures; and (3) to maintain the muscles moving the hip-joint in as good a tone as possible and to prevent contractures occurring in these muscles. These objectives can best be achieved by progressive exercise therapy carried out with the help of a Guthrie Smith frame, or as a variant in a hydrotherapy pool. It should be impressed on the patient that home exercises play an equally important part in his treatment, and that exercises in the department are a prelude to self-treatment.

Heat has played a much overrated traditional role in the therapy of osteoarthritis of the hip, and it seems likely that its main role is to relax the muscles around the joint prior to exercise therapy. In this connexion short-wave diathermy with its deeper heating is probably the most effective agent.

Relief of pain by analgesics should always be attempted, and in this field tab. codein. co. is the most effective of the commonly used compounds. Dramatic relief can sometimes be obtained with phenylbutazone provided no contra-indications exist, but dosage should not exceed 600 mg. a day, and frequently a reasonable result can be obtained on a dose of 300 mg. a day. If sensitivity to the drug is to occur, it usually is manifest in the first few weeks of treatment, and after this period the probabilities of a reaction are small. Many forms of intra-articular therapy have been advocated in the past, but most have now been abandoned except for the use of hydrocortisone, which may occasionally produce some relief. Radiotherapy also is now rarely used, though it too is

worth a trial in patients who are unfit for more radical procedures. Manipulation of the hip-joint has also become unfashionable, but in experienced hands and using the minimum of force temporary benefit can be obtained, presumably as this manœuvre maintains the capsule in a stretched state and so increases the weight-bearing area.

Surgical Treatment

The main indications for surgery are: (1) increasing pain unrelieved by any of the methods already described; and (2) the necessity for a pain-free, stable joint, especially in the younger patient whose occupation requires him to undertake heavy work. The present position of surgery in osteoarthritis of the hip-joint has recently been fully reviewed both by Charnley (1960) and by Ring (1960). Charnley discusses the four basic surgical procedures of arthroplasty, arthrodesis, osteotomy, and pseudarthrosis. Theoretically, the ideal surgical result should be a mobile, pain-free, stable joint. However, the high hopes derived from reconstructive procedures such as the Smith-Petersen cup operation and later the Judet operation have failed to be realized, although in 5% to 10% of patients good results have been obtained by arthroplasty over a number of years. In view of this small percentage of long-term successes, and the need for re-operation where a mechanical failure has occurred, arthroplasty has been largely abandoned by most surgeons as a routine procedure. Newer prostheses and methods continue to be tried, and it is to be hoped that a satisfactory method will ultimately be evolved.

Arthrodesis of the hip has not always been a popular operation, owing to the occurrence of unreliable bony union, stiff knees following prolonged immobilization in plaster, and backache secondary to the excessive strain placed on the lumbar spine. Watson-Jones (Watson-Jones and Robinson, 1956), in a survey of 141 cases operated on by himself, reports sound bony fusion in 94% and the recovery of painless knee movements in 91% of cases. He considered that low back pain arose from strain caused by the hip being arthrodesed in a deformed position. The advantage of a successful arthrodesis is a sound, pain-free limb, and it is therefore the operation of choice in the younger age-groups with unilateral disease who have a good range of knee movement on the affected side. The disadvantages, apart from its occasional failure, are the long period of convalescence involved and the difficulty that some patients with a stiff hip have of sitting down and going to the toilet, and also the undue strain thrown on the other hip, which, of course, could not also be treated by an arthrodesis.

In the older age-groups considerable relief of symptoms can be obtained by an intertrochanteric osteotomy of the McMurray type. This procedure allows the retention of some degree of mobility in the joint and can be used in

cases of bilateral disease; also with modern methods of internal fixation convalescence is short and the risk of post-operative complications diminished. Lloyd Roberts believes that the relief of pain following this operation is due to the diminution of capsular tension, provided that the distal fragment of the femur is in a good functional position. Other surgical techniques, such as variations of McMurray's operation and pseudarthrosis, have been developed and give good results in various individual surgeons' hands, but at present arthrodesis and McMurray's osteotomy remain the two standard surgical procedures.

When all else fails or in patients unsuitable for surgery, neurectomy may be tried, and probably as a last resort a weight-bearing calliper may bring the unfortunate sufferer some relief.

ACKNOWLEDGMENTS

I should like to thank Professor Trueta for permission to reproduce Figs. 1–3 on Plate XXIII, and Mr. G. Lloyd Roberts for Figs. 1 and 2 on Plate XXIV; also the Editor of the *Journal of Bone and Joint Surgery* for his permission for their reproduction.

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REVIEWS OF BOOKS

BACK PAIN. DIAGNOSIS AND TREATMENT. By J. McM. Mennell. *Pp. 226. 68s. London: Churchill. 1960.*

In this book on back pain Mennell describes in some detail the movement that may take place at the synovial joints of the spine and the manipulative methods he employs to test their individual range of mobility. The therapeutic manipulations described are an extension of the diagnostic routine, designed to achieve a full range of movements at the synovial joints. In the later part of the book prophylactic advice is given, and other conditions which may cause back pain are sketchily reviewed.

In contradistinction to modern thought, Dr. Mennell attributes most causes of back pain to dysfunction of the spinal synovial joints and recommends manipulative treatment. Some of his statements give a false impression, such as those about intra-articular injections on pages 26 and 27, and those on localization on pages 51 and 56. On page 177 he states that manipulation has no part in the primary treatment of disk lesions, whereas to-day there appears to be mounting evidence that in proper hands this can be a most helpful procedure.

References to the literature are almost absent, and no figures are produced to substantiate Dr. Mennell's claims. The style of writing is often difficult to understand, and this book could not be recommended to undergraduates or those studying for the Diploma in Physical Medicine. To more experienced physicians it may produce food for thought, as much that is said is controversial.

B. G. B. CHRISTIE

AN APPROACH TO OCCUPATIONAL THERAPY. By Mary S. Jones. *Pp. 245. 42s. London: Butterworth. 1960.*

This book is intended to present a survey of the methods used in the occupational therapy department of Farnham Park Recuperative Home, where the emphasis has been on the rehabilitation of the short-term case. Its author, Mrs. Jones, is fortunate in having a combined qualification in occupational therapy and physiotherapy, and throughout the book the emphasis is not so much on occupation as on the therapy most likely to improve function.

The first part deals with the organization of the department, its equipment, and its expected function. This is illustrated by many good-quality line diagrams and photographs. The second part discusses particular disabilities and the best form of therapy for them. The large number of tables, which give not only the number of patients treated, but details of age, sex, and sometimes symptoms, are unnecessary, as no correlation between these facts and therapy is attempted. The number of case histories could well be reduced. One is also surprised to read that boredom and cramp may have contributed to the development of a peptic ulcer in a patient with ankylosing spondylitis, and that the desire to get to the lavatory in private is partly responsible for the more rapid recovery of leg than arm function in a hemiplegic. These, however, are minor points, and the sections on therapy after meniscectomy and amputation are good.

This is undoubtedly the best available work on the occupational therapy useful for rehabilitation, and as such can be strongly recommended.

J. M. EVANS

ABSTRACTS OF THE LITERATURE

Gold Therapy in Rheumatoid Arthritis. Research Sub-Committee of the Empire Rheumatism Council. *Ann. rheum. Dis.*, 1960, **19**, 95.

This is the report of a multi-centre "double-blind" controlled trial. The 200 patients with early active disease taking part were divided equally into two groups, balanced for sex, age, and duration of disease. Patients in the treated group were given a total of 1 g. sodium aurothiomalate weekly by intramuscular injection over a period of 20 weeks. Those in the control group were given 0·01 mg. over the same span. Supportive measures—for example, physiotherapy—were used in both groups, but no other specific medication was given. The trial was designed to permit comparison of the two groups as regards: (1) clinical and functional assessment at 1, 3, 6, 12, 18, and 30 months after treatment was started; (2) radiological change over 18 and 30 months; (3) numbers who had to be withdrawn or for whom a change of treatment became essential; and (4) the incidence of complications.

The gold-treated patients improved by a greater degree than the controls from the third month onwards, and the improvement was apparent 12 months after the last injection was given. Toxic effects, mainly dermatitis, were more than twice as common in the gold-treated group. Gold therapy improved the average clinical condition of the majority over a period of 18 months, but no such advantage was demonstrated radiologically.

MAURICE F. HART

Articular Manifestations of Erythema Nodosum. L. H. TRUELOVE. *Ann. rheum. Dis.*, 1960, **19**, 174.

The literature relating to erythema nodosum with arthritic manifestations is reviewed, and 41 cases seen over a period of 10 years are described.

The average age of patients with bilateral enlargement of hilar lymph-nodes was 32 years; the average age of those without such enlargement was 39 years. There were only five men in the series. Symptoms consisted of pain and stiffness, especially in the morning, knees, ankles, wrists, fingers, shoulders, elbows, and hips being involved. The duration of symptoms was variable, extending from two weeks to four years, but the condition was essentially benign. The erythrocyte sedimentation rate was high initially, and fell in most instances after 16 weeks, but the relationship between the duration of raised E.S.R. and symptoms varied. The patients had typical skin lesions over the legs (arms also in 7 cases) lasting from three days to six weeks. No evidence was found to suggest that rheumatic fever, tuberculosis, or progressive sarcoidosis occurs in these cases.

MAURICE F. HART

Gout, Familial Hyperuricaemia and Renal Disease. H. DUNCAN and A. ST.J. DIXON. *Quart. J. Med.*, 1960, **113**, 127.

The authors investigated the family of a 19-year-old male admitted with renal hypertension and mild gout. Both parents and all surviving siblings also had hyperuricaemia; all but two had renal disease, but none had gout. They conclude that the hyperuricaemia *per se* was the nephrotoxic agent, and suggest the term "hyperuricaemic nephropathy". A further case of renal damage accompanying the hyperuricaemia of polycythaemia vera is reported. Some improvement in renal function has followed benamid therapy.

J. M. EVANS

On the Differential Diagnosis of Paget's Disease. F. CAMPBELL GOLDING. *J. Bone Jt Surg.*, 1960, **42B**, 641.

The author of this paper states that radiological diagnosis of Paget's disease is often easy. Lesions causing difficulty in differentiation are osteoplastic metastases, Hodgkin's disease, mast-cell infiltration, and fibrosarcoma. The lesions of Hodgkin's disease cause difficulty only when vertebrae are involved. Assistance in diagnosis can often be obtained by observing whether the transverse and spinous processes are affected, this seldom happening in Hodgkin's disease. The osseous changes may be the first and only manifestation of Hodgkin's disease for several years. Mast-cell infiltration of bones is extremely rare. A clue to the diagnosis is the presence of urticaria pigmentosa. In fibrosarcoma the bones are not quite so dense as in Paget's disease and patients are younger than might be expected.

J. J. P. LOMAS

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GENERAL INDEX

Abstracts of the literature, 35, 71, 111, 148, 184, 222, 288
Accidents in department of physical medicine, 156
Actinotherapy and electrotherapy, review of book on, 221
Alkaptonuria, 228
AMICK, L. D., and HICKLEY, R. P.: Abbreviated electrodiagnostic tests of denervation, 48
Animals, arthritis in, 163
Antibody production in rheumatoid arthritis, 258
ANSELL, BARBARA M.: Current trends in management of rheumatoid arthritis, 97
Arthritis in animals, 163
——, psoriasis and, 12
——, rheumatoid, antibody production in, 258
——, ——, chloroquine treatment, 88
——, ——, current trends in management, 97
——, ——, 6-α-methyl prednisolone treatment, 122
——, ——, mucoprotein levels and other serological reactions in, 113
——, ——, rehabilitation and resettlement in, 194
——, suppurative, 214

Back pain, diagnosis and treatment, review of book on, 327
BARR, MOLLIE, *see* Greenwood, R., joint-author
BAUWENS, P.: Electrodiagnostic definition of site and nature of peripheral nerve lesions, 149
Biopsy, needle, of vertebral bodies, 236
BREWERTON, D. A., and MANNING, C. W.: Suppurative arthritis, 214
British Association of Physical Medicine, 1959 Annual Meeting, 105
—— —— —— —— ——, 1960 Annual Meeting, 282
—— —— —— —— ——, joint meeting with Heberden Society, 141
—— —— —— —— ——, provincial meeting, Droitwich (1958), 29
BUCHAN, J. F.: Chloroquine in rheumatoid arthritis, 88

Calcaneus, stress fracture of, 211
CALNAN, C. D.: Psoriasis and arthritis, 12
Cervical spondylosis, pilot therapeutic trial, 37
Chair, tilt, for disabled, 213
Chest diseases, physiotherapy in, review of book on, 33
Child, handicapped, review of book on, 146
Chloroquine in rheumatoid arthritis, 88
CHRISTIE, B. G. B., and COOMES, E. N.: Normal variation of nerve conduction in three peripheral nerves, 303
Congress, Third International, of Physical Medicine, programme, 147
COOMES, E. N.: Intensity-duration curves and terminal neuronal reinnervation of muscle, 243
——, *see also* Christie, B. G. B., joint-author
Cripples, International Society for Welfare of, film award, 183

DAWSON, J. E.: Arthritis in animals, 163
Denervation, abbreviated electrodiagnostic tests, 48
Devonshire Royal Hospital, Buxton, centenary meeting, 141
Disabled, tilt chair for, 213
DOBNEY, G. H., and FAREWELL, T.: Useful polythene splints, 57
—— ——: Shoulder adduction splint, 96
Drainage, postural, and respiratory control, review of book on, 146

Elderly, rehabilitation of, 170
Electricity and ultraviolet rays, therapeutic uses, review of book on, 182
Electrodiagnostic definition of site and nature of peripheral nerve lesions, 149
—— tests, abbreviated, of denervation, 48
Electromyogram, muscle force in relation to, 187
Electromyographic changes in hyperkalaemic familial periodic paralysis, 153
Electromyography of postural muscles, 185
——, review of book on, 220
Electronics, physics and, in physical medicine, review of book on, 286
Electrotherapy and actinotherapy, review of book on, 221
ENGEL, C. E.: Sequence still photographs for analysis of movement, 192
Epoxy resins in splint-making, 10
Exercise, progressive, in rehabilitation and physical education, review of book on, 69

FAREWELL, T., *see* Dobney, G. H., joint-author
FELDMAN, CHARLOTTE, HESS, EVELYN V., and MARKHAM, R. E.: Value of mucoprotein levels and other serological reactions in rheumatoid arthritis, 113
Fibula and tibia fractures, effects of delay in rehabilitation, 203
—— —— ——, malleolar fractures, rehabilitation of, 128
—— —— —— shaft fractures, rehabilitation of, 73
Foot and heel pain, 58
FOX, Sir FRANK, notice of death, 224
Fractures and orthopaedics, review of book on, 146
—— of tibia and fibula, *see* Fibula
——, stress, of calcaneus, 211

GLANVILLE, H. J.: Use of epoxy resins in splint-making, 10
GLYNN, J. H., and IRVING, MARY: Laboratory investigations in diagnosis of rheumatic diseases, 1
Gout, 229
GREENWOOD, R., and BARR, MOLLIE: Circulating antibody production in rheumatoid arthritis, 258
Growing up in Newcastle upon Tyne, review of book on, 286

Hand, disabled, rehabilitation of, review of book on, 70
——, peritendinitis calcarea affecting, 94
——, polythene splint for, 57
Handicapped and their rehabilitation, review of book on, 33
——, tilt chair for, 213
HARRIS, R.: Accidents in a department of physical medicine, 156
——: Results of rehabilitation and resettlement in rheumatoid arthritis, 194
——: Simple tilt chair, 213
—— and SARKAR, S. K.: Method for increasing depth heating effect of infrared radiation, 252
Heberden Society, joint meeting with British Association of Physical Medicine, 141
Heel and foot pain, 58
HESS, EVELYN V., *see* Feldman, Charlotte, joint-author
HICKEY, R. P., *see* Amick, L. D., joint author
Hip-joint, osteoarthritis of, 318
Hyperkalaemic familial periodic paralysis, electromyographic changes in, 153

Infrared radiation, method for increasing depth heating effect, 252
Intensity–duration curves and terminal neuronal reinnervation of muscle, 243
IRVING, MARY, *see* Glyn, J. H., joint-author

JACOBS, J. H.: Osteoarthritis of hip-joint, 318
Joint disease, metabolic, 227
JOSEPH, J.: Electromyography of postural muscles, 185

KENDALL, P. HUME: Needle biopsy of vertebral bodies, 236
Knee, flexion contractures, polythene splint for, 57
Kovacs prize, 145, 285

Laboratory investigations in diagnosis of rheumatic diseases, 1
Locomotor system, metabolic diseases affecting, 227, 289
LOMAS, J. J. P.: Peritendinitis calcarea affecting hand, 94

MANNING, C. W., *see* Brewerton, D. A., joint-author
MARKHAM, R. L., *see* Feldman, Charlotte, joint-author
MASON, R. M.: Metabolic joint disease, 227
——, *see also* Steinberg, V. L., joint-author
MATTINGLY, S.: Osteoporosis and osteomalacia, 289
MATTINGLY, S.: Pain in shoulder, 266
——: Psoriasis and arthritis, 21
——: Stress fracture of calcaneus, 211
Metabolic diseases affecting locomotor system, 227, 289
6-α-Methyl prednisolone in rheumatoid arthritis, 122
MORRISON, J. B.: Electromyographic changes in hyperkalaemic familial periodic paralysis, 153
Movement analysis by sequence still photographs, 192
——, graphic representation of, 185, 187, 192
Mucoprotein levels and other serological reactions in rheumatoid arthritis, 113
Multi-centre research project, 285
Muscle force and electromyogram, relation between, 187
——, terminal neuronal reinnervation of, intensity–duration curves and, 243

Neck pain presenting difficulty in diagnosis, 168
Needle biopsy of vertebral bodies, 236
Nerve, peripheral, conduction, normal variation of, 303
——, ——, injuries, review of book on, 68
——, ——, lesions, electrodiagnostic definition of site and nature, 149
Neuronal reinnervation, terminal, of muscle, intensity–duration curves and, 243
Neuroticism in out-patients attending physical medicine and orthopaedic departments, 310
NICHOLS, P. J. R., and PARISH, J. G.: Rehabilitation of malleolar fractures of tibia and fibula, 128
—— ——: Rehabilitation of fractures of shafts of tibia and fibula, 73
NIGHTINGALE, A.: Relation between muscle force and electromyogram in stand-at-ease position, 187
North and Midlands Physical Medicine Club, reports of meetings, 110, 257

Occupational therapy, review of book on, 327
Old age, rehabilitation in, 170
Orthopaedic medicine, review of book on, 68
Orthopaedics and fractures, review of book on, 146
—— in general practice, review of book on, 70
Osteoarthritis of hip-joint, 318
Osteopathic technique, review of book on, 220
Osteoporosis and osteomalacia, 289

Pain in back, review of book on, 327
—— —— foot and heel, 58
—— —— neck presenting difficulty in diagnosis, 168
—— —— shoulder, 266
Paralysis, hyperkalaemic familial periodic, electromyographic changes in, 153
PARISH, J. G.: Effects of delay in rehabilitation of fractures of tibia and fibula, 203
——, *see* Nichols, P. J. R., joint-author
Peripheral nerve conduction, normal variation of, 303
—— —— injuries, review of book on, 68
Peritendinitis calcarea affecting the hand, 94
Photographs, sequence still, for analysis of movement, 192

Physical Medicine, British Association of, *see* British Association of Physical Medicine
—— —— Club, North and Midlands, reports of meetings, 110, 257
—— —— Congress, Third International, programme, 147
—— —— department, accidents in, 156
—— ——, physics and electronics in, review of book on, 286
Physics and electronics in physical medicine, review of book on, 286
Physiotherapy in chest diseases, review of book on, 33
—— —— surgical conditions, review of book on, 69
Polythene splints, 57
Postural muscles, electromyography of, 185
Prednisolone, *see* 6-α-Methyl prednisolone
Psoriasis and arthritis, 12

Radiation, infrared, method for increasing depth heating effect, 252
Rehabilitation after illness and accident, review of book on, 34
—— and resettlement in rheumatoid arthritis, 194
—— centre planning, review of book on, 287
——, delayed, in fractures of tibia and fibula, 203
—— of elderly, 170
—— —— hand, review of book on, 70
—— —— handicapped, review of book on, 33
—— —— malleolar fractures of tibia and fibula, 128
—— —— tibia and fibula shaft fractures, 73
——, progressive exercises in, review of book on, 69
Research project, multi-centre, 285
Resins, epoxy, in splint-making, 10
Respiratory control, postural drainage and, review of book on, 146
Review series, 12, 58, 97, 170, 214, 266, 318
Reviews of books, 33, 68, 146, 182, 220, 327
Rheumatic diseases, laboratory investigations in diagnosis, 1
RICHARDSON, A. T.: Pain in foot and heel, 58

SAINSBURY, P.: Neuroticism in unselected outpatients attending physical medicine and orthopaedic departments, 310
SARKAR, S. K., *see* Harris, R., joint-author
Shoulder abduction splint, 96
—— pain, 266
Soft-tissue lesions, diagnosis of, review of book on, 68
Spine, *see also* Vertebrae
——, diseases of, review of book on, 182
Splint for shoulder abduction, 96
Splints, epoxy resin, 10
——, polythene, 57
Spondylosis, cervical, pilot therapeutic trial, 37
STEINBERG, V. L., and MASON, R. M.: Cervical spondylosis—pilot therapeutic trial, 37

THOMPSON, M.: 6-α-Methyl prednisolone in treatment of rheumatoid arthritis, 122
Tibia and fibula fractures, *see* Fibula

Ultraviolet rays and electricity, therapeutic uses, review of book on, 182

Vertebrae, needle biopsy of, 236

WARREN, MARJORY W.: Rehabilitation of elderly patients, 170
WILLIAMS, I.: Two cases of neck pain presenting difficulty in diagnosis, 168
WRIGHT, V.: Psoriasis and arthritis, 17
Wrist, polythene splint for, 57

Xanthomatosis, 233

ABSTRACTS SUBJECT INDEX

Anaesthesia, epidural, neurological complications, 72
——, ——, paraplegia after, 72
——, local, recent advances, 36
Antibody response to booster dose of poliomyelitis vaccine, 148
Arteries, obstructed, low back pain due to, 222
Arteritis, temporal, phenylbutazone treatment, 72
Arthritis and chronic rheumatism, advances in treatment, 222
—— associated with ulcerative colitis, 35
——, rheumatoid, corticotrophin treatment, 222
——, ——, gold therapy, 328
——, ——, Reiter's disease, and ankylosing spondylitis, comparative X-ray study, 111
——, ——, sanatorium treatment, 184
——, ——, treated with corticosteroids, incidence of peptic ulcer, 111
Arthrodesis, bone-peg, of first metatarsophalangeal joint, 224

Back pain, low, due to arterial obstruction, 222
Back, pain, low, pathology, 222
Beat-knee, clinical study, 112
Blood flow in forearm during exercise, 224
Brachial plexus, traction lesions, prognosis, 112

Carcinoma, neurological syndromes associated with, 71
Chondromalacia of patella, 288
Colitis, ulcerative, arthritis associated with, 35
Collapse, surgical, during and after corticosteroid therapy, 72
Conduction velocity of motor nerve fibres to hand and foot muscles, 288
Corticosteroid therapy of rheumatoid arthritis, incidence of peptic ulcer, 111
—— ——, surgical collapse during and after, 72
Corticotrophin in rheumatoid arthritis, 222
Craniopathy, metabolic, aetiology, 111

Deformity in chronic rheumatic diseases, "durafoam" splints to prevent, 36
"Durafoam", new material for rest splints, 36
Dystrophy, reflex sympathetic, 36

Electrodiagnostic stimulators, 35
Erythema nodosum, articular manifestations, 328
Exercise, blood flow in forearm during, 224

Facial nerve section, electrical changes after, 288
Fingers, familial osteoarthropathy of, 71
Foot, multiple ossification centres in epiphyses of long bones, 223
Forearm, blood flow during exercise, 224
Fractures of tibial shaft, disabilities after, 36
—— —— —— ——, speed of healing after, 36

Gold therapy in rheumatoid arthritis, 328
Goniometer designed for hand, 112
Gout, familial hyperuricaemia, and renal disease, 328
—— in negro females, 111

Hand goniometer, 112
——, multiple ossification centres in epiphyses of long bones, 223
Hydrocortisone, intra-articular, in high concentration, 35
Hyperostosis cranii, aetiology, 111
Hyperuricaemia, gout, and renal disease, 328

Joint manifestations of erythema nodosum, 328

Kidney disease, gout, and familial hyperuricaemia, 328
Knee osteoarthritis, value of intra-articular injections in, 72

Lumbo-sacral spine, "normal", 184
Lupus erythematosus phenomenon, mechanism, 148
—— ——, systemic, 71
—— ——, ——, clinical diagnosis, 71

Meralgia paraesthetica, 223
Metatarso-phalangeal joint, bone-peg arthrodesis of, 224
Muscles of hand and foot, conduction velocity of motor nerve fibres to, 288
Myopathy, thyrotoxic and polymyositic, in aged, 111

Nerve motor fibres to hand and foot muscles, conduction velocity, 288
——, peripheral, lesions, sensory nerve action potentials in, 288
Neurological complications of epidural anaesthesia, 72
—— syndromes associated with carcinoma, 71

Occupational therapy, evaluation of psychological effects, 224
Old age, thyrotoxic myopathy and polymyositis in, 111
Ossification centres, multiple, in epiphyses of long bones of hand and foot, 223
Osteitis ilii condensans, prognosis, 224
Osteoarthritis, cervical, surgical treatment, 222
—— of knee, value of intra-articular injections in, 72
Osteoarthropathy, familial, of fingers, 71

Paget's disease, differential diagnosis, 328
Pain, congenital insensibility to, 148
—— in shoulder and arm, aetiology, 223
—— —— —— —— —— from neurological involvement, 223
——, low back, due to arterial obstruction, 222
——, —— ——, pathology, 222
Paraplegia after epidural anaesthesia, 72
Patella, chondromalacia of, 288
Peptic ulcer in rheumatoid arthritis treated with corticosteroids, 111
—— ——, prednisolone therapy and, 35
Phenylbutazone treatment of temporal arteritis, 72
Poliomyelitis, diagnostic problems, 148
——, epidemiology, 148
——, management, 148
—— vaccine, booster dose, antibody response to, 148
—— ——, formalin-killed, present situation, 148
Prednisolone therapy, peptic ulcer and, 35
Psychological effects of occupational therapy, evaluation, 224

Quadriplegia in teenagers, rehabilitation, 223
de Quervain's disease, 36

Rehabilitation, advances in, 184
—— of quadriplegic teenagers, 223

Reiter's disease, rheumatoid arthritis, and ankylosing spondylitis, comparative X-ray study, 111
Rheumatic diseases, chronic, "durafoam" for rest splints to prevent deformity, 36
Rheumatism, chronic, and arthritis, advances in treatment, 222
Rheumatoid factor, isolation and characterization, 184

Sanatorium treatment of rheumatoid arthritis, 184
Sensory nerve action potentials in patients with peripheral nerve lesions, 288
Shoulder and arm pain, aetiology, 223.
—— —— —— —— from neurological involvement, 223
Shoulder–arm syndrome, basic concepts, 223
Spine, lumbo-sacral, "normal", 184
Spondylitis, ankylosing, clinical features and course, 35
——, ——, Reiter's disease, and rheumatoid arthritis, comparative X-ray study, 111
Stimulators, electrodiagnostic, 35

Tibia shaft fractures, disabilities after, 36
—— —— ——, speed of healing after, 36
Traction lesions of brachial plexus, prognosis, 112
Trochanteric syndrome, 71

Ulcer, peptic, in rheumatoid arthritis treated with corticosteroids, 111
——, ——, prednisolone therapy and, 35

Vaccine, poliomyelitis, booster dose, antibody response to, 148
——, ——, formalin-killed, present situation, 148

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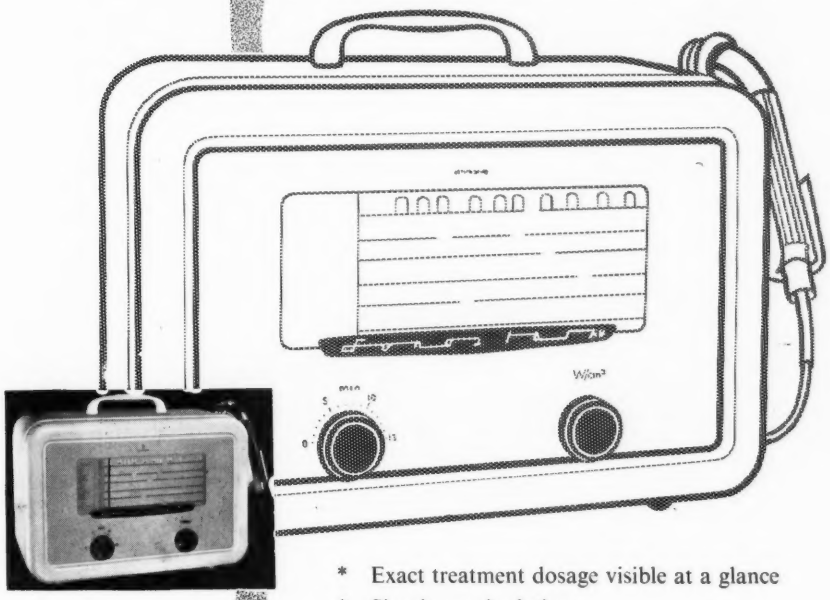

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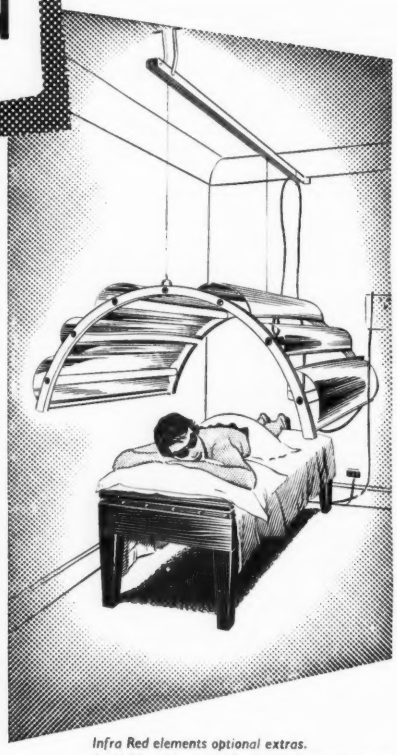

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Contents

Original Papers

METABOLIC DISEASES AFFECTING THE LOCOMOTOR SYSTEM

II. Osteoporosis and Osteomalacia. STEPHEN MATTINGLY 289

NORMAL VARIATION OF NERVE CONDUCTION IN THREE PERIPHERAL NERVES 303

B. G. B. CHRISTIE and E. N. COOMES

NEUROTICISM IN UNSELECTED OUT-PATIENTS ATTENDING PHYSICAL MEDICINE AND ORTHOPAEDIC DEPARTMENTS 310

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